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THE PEDIATRICIAN TODAY AND TOMORROW

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MEDICINE is one of the oldest professions. It has passed through all the periods of history, those of enlightenment and of darkness, without ceasing to make progress. There is every reason to believe that we will not be displaced at present or in the future. Our knowledge regarding health and disease is at the highest point in history. We have much reason to be proud of the status of medicine in America today, both from the standpoint of a science and a practice.

Although pediatrics, as a specialty, is of recent origin, it has, for the past twenty-five years, shown an extraordinary advance in growth and importance. The number of men who have entered this field of medical work has increased alarmingly during this time. Dr. Veeder in a recent address informed us that pediatrics as a special field has increased at a rate far beyond the increase of specialization as a whole. Today one out of every ninety physicians, according to the *Directory of the American Medical Association,* is limiting his practice to pediatrics.

But these men, after a long and expensive training, are confronted as they enter their careers with the following changing conditions, each and all of which have a direct bearing on their effectiveness, their ability to live, their ability to rear families properly, and at the same time find stimulation and joy in their work:

(1) A decreasing birth rate; (2) a decrease in infant and childhood morbidity, due to the increasing use of modern preventive measures; (3) a decreasing number of individuals able to pay even moderate fees for service; (4) extension and improvement in public health activities; (5) a wide dissemination of knowledge concerning the physical and mental hygiene of childhood; and (6) overproduction of pediatricians.

Can we be instrumental in decreasing the number of men who, when confronted with the actualities of practice, will be and are being faced

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with disappointment, often with loss of ideals and loss of morale? Remember that fully 80 to 85 per cent of all pediatrically trained men enter practice. I say, "Yes," if we are willing to take our part in the social economic life of today and do not confine ourselves to medicine as a science and an art but also endeavor to influence public thought and attitude to a sane and realistic conception of the medical man's duties and contributions to the social world.

The world is moving very rapidly these days. One can almost say what was new today is old tomorrow. We are learning as never before to change our viewpoints and ideas. People in many vocations, willingly or unwillingly, are being forced into action. The complexities of modern life and the pace at which we live have increased the demand on thought without providing us with a corresponding increase in the faculties with which we may reach sound judgments; therefore we must be alert to keep our thinking from being clouded by emotions, usages, and unwarranted or unsound traditions.

Tradition and idealism have, properly, always been a part of medicine. Our habit trends and training tend to make us individualistic and conservative. The very fact that we are interested in a science makes us cautious and skeptical until we have ample proof that a thing is right. We are taught to reason, to evaluate findings carefully, to utilize instruments of precision, and then finally to reach a conclusion, and, when we have done all this, the pathologist at the autopsy table demonstrates our mistakes.

How fortunate we are in comparison with those in other walks of life, those for instance who conduct us in our religious life, in our civic and in our economic life, and those who promulgate our laws and govern us. Here many mistakes are made, but unfortunately the true cause of a mistake frequently cannot be determined even after we have felt its effects. While logical reasoning and actual proof are fundamental to medicine, when we enter the social field of medicine, a certain degree of imaginative thinking and vision is essential. The pediatrician as he is constantly dealing with the young unfinished human being is already accustomed to rapid changes. They are nothing new to him; they are part of his daily life; therefore it should be easier for the pediatrician to adjust himself.

The question is, What course is he going to take? He can follow either of two courses: he can wait until the layman shows him how he must proceed, or he can help evolve a plan. If he follows the latter method he can, by sincere cooperation and understanding and by educating and directing lay opinion, become a leader of most of the worthwhile movements. Once we admit that all these conditions confront us and that they are of a nature which threatens the future welfare of the

profession as well as those whom we serve, it seems inevitable that we should depart somewhat from the traditional and expedient and meet the problems in a sane and realistic fashion.

Some of the problems which confront us today and which are of the most importance to the profession as a whole do not bear heavily on those of us who have long since served our apprenticeship, but they are so vital to the younger man as to threaten the integrity of his continued existence as a pediatrician. Nor can he judiciously or expediently speak for himself. The moment he becomes articulate he is accused of being self-seeking, of disloyalty to medical ideals, and he is branded as mercenary and revolutionary.

We all believe that if possible every one should receive good medical care. The medical profession has always rendered free unselfish assistance to those in need. It has been almost an unwritten law that the physician should respond to such calls for help without thought of reward. Our mental habits and ideas are shaped according to the age in which we live. In past centuries, due to conditions and ideas different from those we now have, this type of so-called charity was considered a virtue and a solution of a portion of the indigent problem. It never really was satisfactory to the physician or the patient and today is even less so. The state does and must assume responsibility for those unable to provide for themselves.

The young pediatrician has spent from nine to twelve years preparing himself for his profession and represents an investment of at least $8,000.00 to $12,000.00, and yet at the present time he is confronted with situations which frequently deny him a sufficient income on which to live. What the future may offer him is still another question, and he will and should be a factor in deciding this part of his problem. The state and every smaller political unit pays every other servant in its employ, from janitor to governor, and even exempts them from some taxes.

The pediatrician soon after his entrance into practice is frequently called upon to render medical social work to his community. He enlists for work in welfare stations, tax-supported hospitals, or other public institutions, such as schools, orphanages, etc., generally without any, or at best a negligible, compensation.

Shall we today penalize the pediatrician for his idealism, of which he has given ample proof in the past? Why should he be treated differently from any other servant of the state? Is it because he is better fitted and able to fill his job than most public servants? Is it because he is an idealist? Is it because he can better afford to give of himself and his time than other citizens? No, it is primarily because of bondage to tradition. We ourselves are at fault. We have educated the public and its officials into the idea that the medical man should and will render

public service without compensation. No matter how tenable this thought was when society was simple, it becomes untenable in our present highly complex industrialized society.

The time has come, I believe, when reeducation is necessary. The pediatric public servant deserves fair remuneration for his work. This will lead to more satisfactory service and a more nearly proper relationship between the medical man and those dependent on public health methods for care. The question of mass service and the extent to which public health methods shall be employed is, however, not solved by compensation; that is merely one step in the right direction.

This thought is not a mercenary one. The quality of service available cannot be greater than the servant is able to give, but the servant is just a human being, and, if he is unrewarded, generally the quality and quantity of the service is less than it could be, and, in addition, the inevitable lack of continuity of service is often disastrous, as has long been evident to you all.

We hear much, especially in medical circles, regarding the encroachment by public health activities upon the field of the practicing pediatrician. This controversial question cannot easily be resolved to simple terms. It cannot be answered by a simple "Yes" or "No." The public is child-minded and rightly so. It wants everything that may add to the welfare of the child. Pediatricians and pediatric organizations have aided and sponsored this movement to extend governmental agencies for child health. However, the laity are dominant in the control of these movements, and it is high time for the pediatrician to take his proper place at the head of the procession.

Unfortunately we too often take an antagonistic arbitrary position. Within the pediatric profession there are many whose opinions differ, in degree only, to be sure, regarding the scope of these enterprises and their character. Naturally then the ideas and viewpoints of the layman will at times be at even greater variance with those of the medical man. To sit about in medical societies, and, what is more common still, in small groups, and deplore and decry the situation among ourselves will be of no great avail and certainly will not help us. In such a situation a spirit of cooperation, a spirit of willingness to exchange ideas, and a spirit of tolerance for opposing convictions or near convictions are necessary to bring about the best solution and develop the best remedies.

It is evident that we must meet lay organizations and public officials and demonstrate to them that we are able to transport ourselves mentally to their viewpoint and that we admit that some of the ideas and conclusions on both sides of these debatable questions are wrong and untenable. This means that we will educate each other, we will both learn that these problems may have many ramifications and possibly immediate, as well as future, aspects as regards the individual and society as a whole.

Finally, in this manner we can and must evolve an answer based upon cooperation and a broad conception of the problem. During these deliberations we should, however, not lose sight of the primary object—the welfare of the child. Ultimately, I believe the laymen and the profession will then decide that all these movements for child betterment require the active guidance and leadership of qualified pediatricians, and now the pediatrician must be ready to respond to the gong of the bell. Welfare and public health agencies, as well as the practicing pediatrician, will then find their proper spheres, will travel peacefully side by side, and the child and his welfare will get the full benefit of pediatric learning and progress. These movements will then be properly manned, guided, and carried on by the only persons understanding child growth and development and its peculiarities in health and disease—the pediatrician.

We have been and are taking part in all the important movements pertaining to child welfare, but frequently we have not kept pace with the momentum gained by these movements and have allowed nonmedical groups to control and lead them, and thus we are, so to speak, left on the outside of the fence peeking in through a knothole. This is well exemplified by an attitude toward the problem of birth control, which has been before us many, many years. How many medical organizations have seen fit or have had the courage to take a stand for or against this measure? Silence is not necessarily always motivated by disapproval. No one is advocating compulsory birth control, but many well-educated, honest, intelligent, conservative people have asked for medical and governmental recognition of birth control methods for those who need or wish them. I feel we are not misstating facts if we say that many a physician in this organization and in every other medical organization has practiced birth control in his own family from the time of his marriage and has advised some of his individual private patients regarding contraceptive procedures. For various reasons some are, however, unwilling publicly to defend and support or emphatically to decry this movement.

What has been the consequence? Almost every newspaper and drug store advertises birth control measures under the guise of feminine hygiene. And instead of being in the hands of the physician and being properly utilized, it is in the hands of the public with no restraining influence and, I fear, probably will remain in their hands. This problem certainly concerns the pediatrician and his work. He is the first one to look after the products of conception; he sees and knows the results, both social and medical, of indiscriminate childbearing. Do we suppose that by evading the issue we avoid the problem or, as a profession, escape the penalties of an attitude that is hardly sincere and certainly is not realistic in meeting a very present issue?

To speak of the human side of medicine is almost a trite expression. We have several times mentioned the progress made in the science of medicine, but we now wish briefly to speak of pediatrics not only as a science, but also as an art and a philosophy. A full discussion is impossible and has already been so ably made in addresses by Dr. Abt and Dr. Brennemann.

Partly because of the prominence given to diagnosis in our medical schools and hospitals, it is only natural that diagnosis should almost dominate at times the pediatrician's attitude and relationship with the parents of his patients. The parents' interest, as it should be, is solely centered on the child. The physician is here to alleviate suffering and illness, but in carrying out his function he must not lose sight of the fact that he must do more than satisfy medicine as a science. He must consider his profession also as an art and a philosophy. He must come to a realization of the human side of medicine. I feel that not infrequently, self-satisfied and proud of an established diagnosis, our subsequent course in our contact with the parents is lacking in sympathetic, responsive understanding. We all know how easy it is to cause unnecessary severe mental suffering in the parent by discussing the patient's illness in an ill-advised manner, by instilling fear, by an inopportune display of our medical knowledge in reciting all the many complications of a disease in order to satisfy our ego and perhaps in order not to assume too much responsibility. Self-interest has no place here. We should lessen fear by wise and thoughtful information as to the real condition of things. The mind and the heart must enter into it, sometimes the one, sometimes the other gaining the ascendancy, depending upon the situation, but rarely is it the mind alone.

The pediatrician, of all medical men, needs to acquire and apply the human side of medicine in his dealings with young parents who are so badly in need of comfort, assurance, and humane treatment. The pediatrician, often endowed with high qualities of mind and heart, already possesses the qualities necessary for such conduct, but while possessing the tools he certainly requires some instruction in their use. Why should not our medical institutions set aside some time in their curriculums for a discussion with the student of this very important branch of medicine? It cannot be taught, but it can be discussed.

Our medical schools, our training of medical men in the hospitals, have greatly improved in the past fifteen to twenty years. We now have a Board of Pediatrics to pass upon the qualifications of the pediatrician; we have this society which soon will admit only certified pediatricians. Pediatrics has made a place, an enviable place, for itself in medicine in this country. The pediatrician as a general rule probably works harder than men in other specialties. He devotes more time and thought to public welfare; he renders more services for comparatively small or moderate fees and, in this era of institutional care, looks after more pa-

tients in the home than other specialists. If he realized it, he is also in a position, because of his intimate contact with the young people, I mean the young married people of this country, to wield a tremendous influence in helping to form their ideas toward medicine and toward conditions of society as a whole. The stage is set for him for leadership as a citizen.

Why should not the pediatrician, for his efforts along preventive medicine and immunology as well as for his curative efforts, receive the same credit and recognition as the surgeon, the laryngologist, and other specialists? Does a hernia operation or any other operative procedure, such as a tonsil extirpation, require more learning, skill, and judgment than the pediatrician gives to a case of dysentery, cholera infantum, endocarditis, or many other pediatric illnesses? Not at all, but I am afraid we feel our youthfulness as a specialty; we still display a little sense of inferiority, or, better said, we have not been aggressive enough and have not demanded the place in medicine which we deserve to occupy.

We have for the first time in this country a national society of pediatricians, this Academy. We are now in a position to act as a national unit. The wise men who conceived this association fortunately did not make it a purely scientific society. We are free to discuss, support, initiate, and put before the public anything pertaining to the welfare of the child. We should cooperate intelligently with all other medical organizations; we should study the many problems of today with them if they concern the good of medicine or humanity, but, if necessary, we should also have the courage of independent thinking and action so that our actions may conform to our intellectual convictions.

I hope this body will remain free of medical politics, a most vicious influence in medical organization. I hope the younger men will always have a voice in its policies and that they will feel that the executive officers and committees welcome and wish to encourage constructive suggestions. I hope that we will always maintain a broad viewpoint and have enough imagination to see the future possibilities and probabilities of any movement and measure we may support or initiate. I hope that, in addition to the stimulation along medical lines which we may afford to our members by our activities and by regional and general meetings, we will also inspire them to take their place in their communities as citizens prepared and ready for leadership.

THE GASTROINTESTINAL PORTAL OF ENTRY IN POLIOMYELITIS

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THE virus of poliomyelitis has almost an obligate affinity for axis cylinders of unmedullated nerves, i.e., naked gray fibers. It may also travel along the axis cylinders of medullated nerves after the nerve has been irritated, but the results are inconstant. Medullation seems to serve as a bar to its spread. It even prevents absorption when virus is placed outside such a nerve.[1] Virus soon disappears after intracerebral injection, and presumably it does not multiply in the white fibered medullated area beneath the cortex. Thus, in some fashion, medullation seems to protect nerves from virus.

It is fair to assume that in the human being absorption occurs naturally only in those places where naked axis cylinders may come in contact with the virus. However, a "take," or the successful production of the disease, depends upon an unbroken connection between the absorbing axis cylinders and the central nervous system. The distance between these points must not be too great, since the virus may be absorbed and excreted before reaching the cord. Thus rapidity of absorption, the speed and ease of transmission, the virulence of the strain, etc., are factors upon which a successful take depends. The places in the body where absorption could occur are in the olfactory area,[2] the chordae tympani,[3] the gastrointestinal tract along vagal[4] or sympathetic fibers,[5] locally from the bladder,[6] and possibly by way of the respiratory nerve fibers. Where the course to the central nervous system is short and most direct, the experimental disease is easily produced. However, the ease with which the disease is produced experimentally is not *ipso facto* proof that the portal of entry used thus to produce the experimental disease is the natural portal of entry in the human being.

In most instances of human, as well as of experimental, poliomyelitis the lower extremities are first affected. Because of this, Wickman and even Harbitz and Scheel first thought that the disease must have its entry through and spread from the gastrointestinal tract. Sicard[7] suggested that the spread to the cord might be by way of the sympathetic system.

If one remembers that the virus of poliomyelitis has an affinity for gray fibers, he is at once struck with the anatomic fact that the dis-

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ease usually attacks the nerves which come from those areas lacking white rami communicantes or medullated connections, i.e., those segments which supply the muscles of the arms and legs. The somatic nerves possess no direct white fibered connections with the sympathetic system in the areas from the second lumbar to the second sacral segment and none upward from the first thoracic segment. Here there are only gray fiber connections, and it is in these segments of the cord that involvement occurs. One can easily understand the spotty spread of the disease should one presage a spread of the virus from the gastrointestinal tract to the cord by way of the sympathetic gray fibers and the somatic nerves of these segments lacking white rami communicantes, i.e., to the cord in the lumbar area. With a greater susceptibility, the spread would be up along the sympathetic collateral chain to the other segments that lack white rami communicantes, i.e., to the cervical area. As the virus spreads, it passes and irritates the spinal ganglion and produces that curious segmental type of pain that precedes somatic

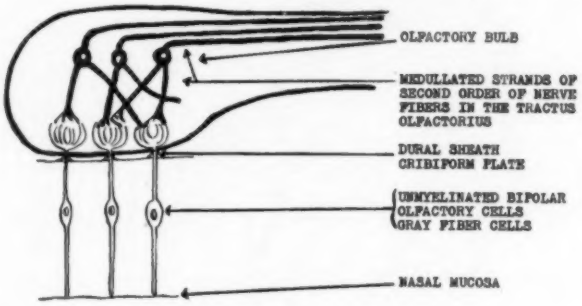
OLFACTORY BULB
MEDULLATED STRANDS OF SECOND ORDER OF NERVE FIBERS IN THE TRACTUS OLFACTORIUS
DURAL SHEATH CRIBIFORM PLATE
UNMYELINATED BIPOLAR OLFACTORY CELLS GRAY FIBER CELLS
NASAL MUCOSA

Fig. 1.—Olfactory area.*

muscular palsies from the same segments. The virus is also absorbed by the end fibers of the vagus nerves; the unmedullated, gray, and postganglionated fibers of these nerves are located only in the wall of the gut. The vagus nerve itself is chiefly nothing more than a set of medullated preganglionic collector fibers, and, since we have seen that medullated nerves are not too easy to affect, it is understandable why all persons with poliomyelitis do not get spreads along both vagi and die because of bulbar palsies. In most instances the virus is not potent enough to bridge the synaptic gap, but occasionally with toxic material it should and does. The isolated palsies of the seventh nerve and the bulbar types of involvement are easily understood if one can picture a spread along the chordae tympani or the vagus nerves.

If in the human being the spread of virus is arrested at places where white fiber interpositions or medullations occur, then there will not

*These diagrams should be studied in connection with those found in this Journal, Vol. 7, pp. 280 and 282, 1935.

be seen much paralysis of these muscles whose innervations come from the second sacral segment and below since white rami communicantes start here again.[6] Usually the patient should be able to move the big toes, even in the most severe case of paralysis. The levator ani, coc-

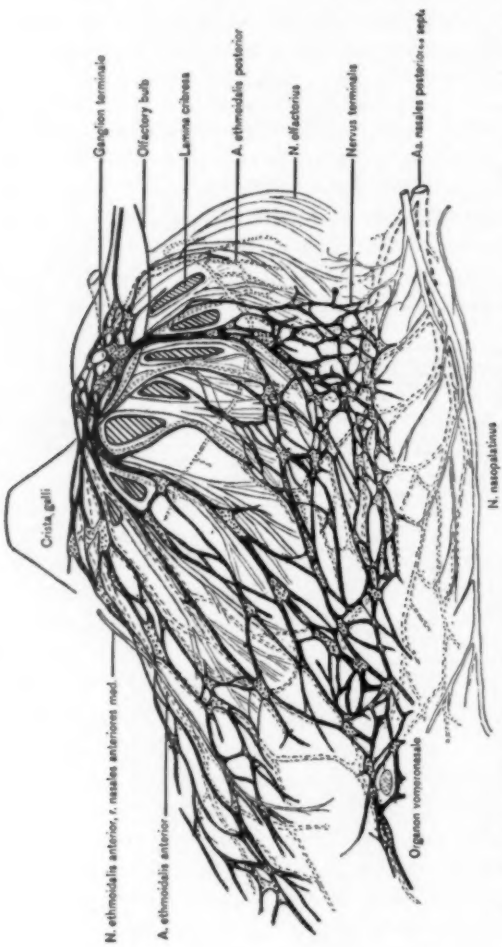

Fig. 2.—Semidiagrammatic representation of the right side of the nervus terminalis of a negro infant, seen from the median plane projected upon the olfactory bulb and fila olfactoria. Ganglion cells represent the location of main aggregations but not the actual number of cells. Rami of the nervus terminalis are made black to stand out from the other structures (×6). This figure is from Brookover, C.: The Peripheral Distribution of the Nervus Terminalis in an Infant, J. Comp. Neurol., Phila. 28: 349-360, 1917.

cygeus, and other perineal muscles should not be affected in the ordinary case. The fingers could likewise be flexed even though the arm muscles should be involved. This is exactly what is seen in the average clinical case.

The gastrointestinal tract supplied by the sympathetic and parasympathetic systems has often been found to be involved before any somatic paralysis has appeared.[6] Equally astonishing is the fact that

the urinary bladder is often found to be paralyzed or involved before any symptoms of somatic paralysis have appeared anywhere.[8] This urinary paralysis could not be central or cord in origin, since in most instances there is no paralysis of the muscles innervated from the same segment. Another curious thing is that this paralysis of the bladder usually disappears within from seven to ten days, irrespective of the massiveness of the somatic lesion elsewhere. Such ready recovery would be impossible were the lesion of cord origin.

In clinical studies I have observed modifications of the abdominal and other reflexes which are best explained as being due to an involvement of the sympathetic system later followed by an irritation of the involved somatic nerve.[9] A like involvement occurs in typhoid fever[10] and occasionally in intestinal influenza. When individuals with paralysis limited to one leg are injected with pilocarpine, sweating starts sooner and persists for a longer time over the skin of the segments of the paralyzed than of the nonparalyzed area.[11] When adrenalin is ad-

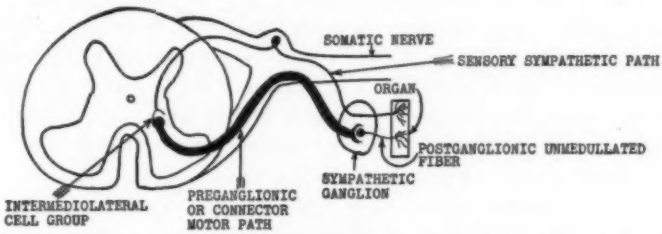

Fig. 3.—Sympathetic arc and connections with spinal cord. Composition of a simple thoracolumbar efferent sympathetic path with visceral afferent connection.

ministered following pilocarpine, in most instances the sweating stops promptly over the unparalyzed, but continues over the paralyzed side, demonstrating that the sympathetic fibers supplying the sweat glands are involved.

The stools of patients ill with poliomyelitis are more toxic for a time after the infection.[12] There is also a definite decrease in serum agglutinins against the ordinary enteric organisms in the blood serum of patients ill with the disease.[13] The disease occurs during the season when perishable foods become ripe.[14] The local drainage glands, the mesenteric, are often found enlarged at autopsy as in typhoid fever. The heights of the spring and fall peak curves occur about the same time as in typhoid fever, a gastrointestinal disease.[15] Finally, many of the early symptoms of the disease are referable to the gastrointestinal system.

When the virus of poliomyelitis (Flexner's M.V. strain) is introduced intracerebrally or intranasally, the monkey gets quadriplegia and usually dies, but this is not the type of poliomyelitis seen in the human being.

If one can produce in the experimental animal the same kind of poliomyelitis that is seen in the human being, it is plausible to suppose that the portal of entry used to produce this condition in the monkey might be the same as that taken by the virus in the human being. Many experimenters have tried and failed to produce the disease by way of the gastrointestinal tract. In the *M. rhesus* monkey, a normally unsusceptible animal, the gray fiber endings in the intestine are protected from contact with poliomyelitis virus by active peristalsis and a mucous coating. It would be difficult for a normal animal to contract the disease by that portal of entry unless the virus could be made to approximate the gray fibers. Accordingly, the abdominal cavities of monkeys were exposed, the small intestines clamped with a pair of intestinal clamps a few inches above the ileocecal valve and in another place about a foot or more above this. A potent virus suspension was then introduced into the isolated portion of the intestinal canal until the gut was ballooned out, and the intestines were kept tense until the pinch reflex had disappeared. Poliomyelitis developed.[16] Since the animals might have regurgitated some virus and thus have infected the olfactory area, the gray fibers were approximated by subserosal injections with the same result. The curious thing is that, when proper doses of poliomyelitis virus are injected subserosally, the disease that develops in monkeys is more like that seen in human beings since only a monoplegic or paraplegic paresis or paralysis may result, in marked contrast to the fulminating quadriplegia seen in the experimental animal after intracerebral injection or intranasal instillation of the virus.

In the experiments just described, the avenue of spread is by way of the sympathetic system. There is a direct communication along the sympathetic fibers which supply the blood vessels from the gut to the intermediolateral cell column area in the cord.[17] That the virus can travel along the sympathetic system to the cord has been shown experimentally.[18]

As in the human being, so also in the *M. rhesus* monkey, the blood serum agglutinin titer for the enteric organisms diminishes to zero as the animal becomes moribund but rebounds if the animal recovers.[13] Like human beings also, the stools of monkeys sick with poliomyelitis contain an enterodepressant or toxic factor not present in the stools of the same animals when they were normal.[19] This toxic factor, which appears in the stools of human beings and the experimental animals, although not a virus, is neutralized by convalescent poliomyelitis serum. It is curious also that the most toxic organ found in the guinea pig after subcutaneous injection of toxic stools was the small intestine.[20] When I injected the cortex of monkeys with virus or enteric toxins, nothing happened for days, but when these two entities were combined and injected, heterolateral paresis or paralysis occurred almost im-

mediately.[21] In addition, the bulbar syndrome, as well as the syndrome of the seventh nerve involvement, had been experimentally demonstrated.[3, 4]

In the latter stages of the disease, the lymphocytes seem to be withdrawn from the circulation; there is a definite leucopenia, both lymphocytes and leucocytes participating.[22] In the preparalytic stage there was an increase in leucocytes and a drop in lymphocytes.

After intracerebral injections or intranasal instillations of the virus there is no histopathologic evidence of the disease in the cord before symptoms start, or at least not before three and usually four days

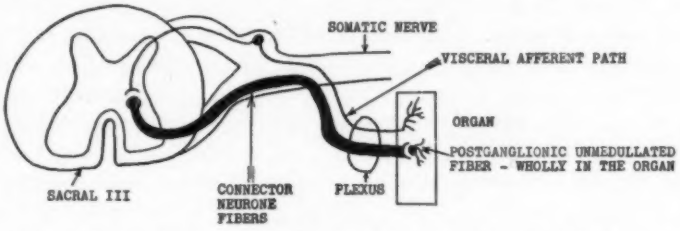

Fig. 4.—Parasympathetic arc (sacral II, etc.) and connections with spinal cord. Composition of an efferent parasympathetic path in the sacral area with visceral afferent connection.

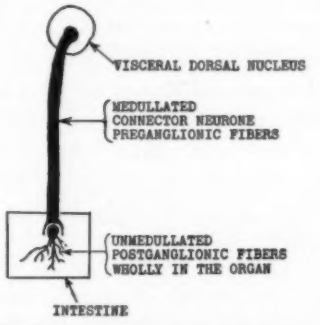
VISCERAL DORSAL NUCLEUS
MEDULLATED CONNECTOR NEURONE PREGANGLIONIC FIBERS
UNMEDULLATED POSTGANGLIONIC FIBERS WHOLLY IN THE ORGAN
INTESTINE

Fig. 5.—Composition of the efferent parasympathetic fibers of the vagus nerve to the intestines. Note the existence of visceral afferent fibers of the vagus nerve in the small and large intestines is questioned. Therefore, they are not shown here, but such fibers can easily be demonstrated elsewhere, as in the esophagus, larynx, etc.

have elapsed after injection.[23] However, when sections of the cords taken from animals in which the virus had been introduced via the gastrointestinal tract are examined, changes can be noted in the lumbar cord area within twenty-four hours after injection.[24] By examination with Nicol prisms, the involvement in the axis cylinders of peripheral nerves can be demonstrated, not in from five to seven days as in the case after intracerebral injection, but within twenty-four hours after gastrointestinal injection.[25] O'Leary and his associates[26] have demonstrated that after intracerebral injections no changes occur in the

somatic nerves until after the anterior horn cells of those segments are destroyed. When the median or sciatic nerves are injected with virus, no injury to the nerve is noted a scant inch beyond the injection; yet when the virus is introduced via the gastrointestinal tract, destruction of the nerve fibers is almost immediate. It is thus obvious that the virus acts differently only when injected via the gastrointestinal nerves.

Römer[27] injected virus intracerebrally, and his animals first got the disease in the legs. Since he could not understand how virus injected intracerebrally could travel though the nervous system and first involve the legs, he concluded that the gastrointestinal tract could not be the portal of entry. It seems agreed by most of those who feel that the olfactory area is the portal of entry in this disease that clinical evidence is of doubtful value. No explanation of the usually earlier and greater paralysis of the lumbar area is offered other than that virus usually involves the anterior horn cells of the lumbar area because of the greater susceptibility of these cells. If the cells of the lumbar area in the monkey have such a high degree of susceptibility, they should respond promptly to local injections of virus into these specially susceptible cells, into the lumbar area itself. There should be no latent period before the disease is produced. But they do not thus respond, and there is a latent period, for when the virus is injected directly into the lumbar enlargement, there is delay before the disease is produced.[1]

The theory of special susceptibility of anterior horn cells of the lumbar area might be more convincing if all cases had involvement in that area or at least first in that area. Why is the cervical enlargement hit later, or, more curious still, why is the bulb solely involved at times? Shall we deduce that in cases with only bulbar involvement or only seventh nerve palsy, the lumbar motor areas of these patients are not specially sensitive and that the involved motor cells are the ones specially sensitive in these instances? How does one explain the way the cervical area comes to be involved later when the virus had already passed through this area to reach the lumbar soil without doing any damage on its way through the cervical enlargement? Must it pass all the way through the central nervous system to get to the lumbar area and then trek back to the cervical and pontine areas to involve these places later or does damage done stay latent everywhere and only evidence itself sooner in the lumbar area? Were that true, then we would expect subsequent involvement of all these areas in every instance of the disease, which does not happen.

It is interesting to note that intranasal inoculation does not initiate the disease if the connection between the gray fibered olfactory nerves and central nervous system is severed. The experimental disease can be introduced via the olfactory fibers, but the disease thus produced

in the monkey is not the usual type of disease that is seen in the human being. Nor do such experiments exclude other portals of entry in this disease.

Poliomyelitis can be given to monkeys if a large amount of virus is injected intravenously.[28] If, however, the olfactory nerve is transected, the disease cannot be given when the intravenous route is used. This does not prove that the olfactory area is the portal of entry in

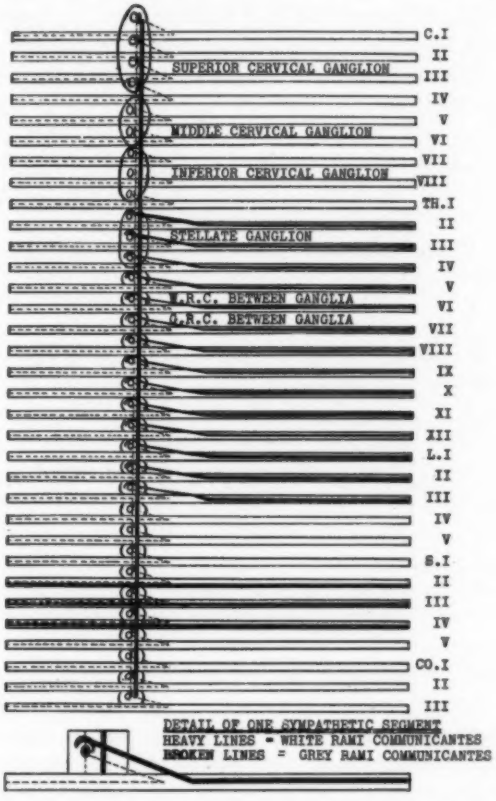

Fig. 6.—Sympathetic nervous system—illustrating areas where white rami occur and where they are absent. (Thoracolumbar outflow—modified after Gaskell.)

the disease. It merely shows that the virus when injected intravenously is excreted among other possible avenues along the gastrointestinal tract, the upper portion or stomodeum end of which has jutting out from and under its mucosal surface the first and thirteenth gray fibered nerves which reabsorb the virus and carry it to the central nervous system.

The virus is not found in the stool contents of the human being; but, if one reflects for a moment, he soon realizes that the virus is absorbed by the gray fibers of the intestine and that days may elapse

before symptoms begin in the human being. Thus it is not strange that we do not find virus in the feces. It would be curious if we did, since any extra material must be evacuated long before the disease becomes manifest.

The virus is never found in the stool contents of the monkey after intracerebral injection! If the virus has an affinity for gray fibers and becomes fixed thereto, why should it be considered logical to expect to find detectable amounts in the stool?

If we assume that the diseases in the monkey and in the human being are both poliomyelitis but with different mechanisms of production, we immediately see how many of the inconsistencies of the disease could be explained. In the monkey the virus itself seems innocuous as judged by the lack of pathologic evidence of its presence between the time of injection and the time when symptoms begin. The disease results in this animal only when the virus spreads down the cord and over the sympathetic system to the gut where it combines with a secondary factor and the combination material destroys motor cells. Only one of these factors is the monkey cord virus that initiates the disease in this animal, innocuous in itself in our opinion, probably a nonliving enzymic catalyst with an obligate affinity for gray fibers and probably similar in physical properties to the mosaic virus.[29] In the monkey the disease is reproduced only when the toxic material produced in the animal's gut can course back over the sympathetic chain and vagus to the lumbar cervical and medullary and pontine areas.[30] Such combining of factors is not new in medicine. In this connection, the experiments of Shope[37] on swine influenza are interesting. He showed that the filtrable virus of swine influenza and *H. influenza suis* act in concert to produce swine influenza and that neither alone is capable of inducing the disease. The secondary rôle of the paratyphoids in Oroya fever may be mentioned.[38]

In the human being both factors may be delivered ready-made or partly so, and absorption along the vagus and sympathetic nerves would give us the usual disease picture. It may be that with certain diseases stasis of the gastrointestinal tract results, and the nerves to the cord are conditioned by enteric toxins so that the disease is produced merely when the virus reaches the cells of the anterior horn. Is there any evidence to bear upon this point? When the gastrointestinal tracts of monkeys are disturbed by any manner of operative interference and some time is allowed to elapse, then heterolateral paresis is produced almost immediately after intracerebral injection.[31] Animals thus operated upon show a marked loss of agglutinins against enteric organisms. When baby monkeys are injected with virus intracerebrally after having had a series of injections of coli enteric toxins and organisms, the production of paralysis is almost immediate.[32] The agglutinin titer of these animals is about nil also.

It is stated that giving large amounts of virus by way of the gastrointestinal tract does not produce the disease in monkeys. Probably it never will unless the monkey is prepared, has a similar constitutional make-up, and is in the same physical condition as is the human being who contracts the disease. If animals could be thus prepared, then the production of the disease should be accelerated. We have seen that baby monkeys injected with coli filtrate and vaccine are predisposed to the disease. We have seen that enteric toxin added to virus enhances its powers of disease production.

Are there states in the human being which naturally have an associated decrease in agglutinin titer against enteric organisms and hence by inference an increase in susceptibility to the virus? We are now able to report that such conditions have been demonstrated.[33] Could we create the same condition in animals? This has been done likewise.[34] What would happen if the animals thus conditioned, as it were, were given the disease via the gastrointestinal tract? The production of the

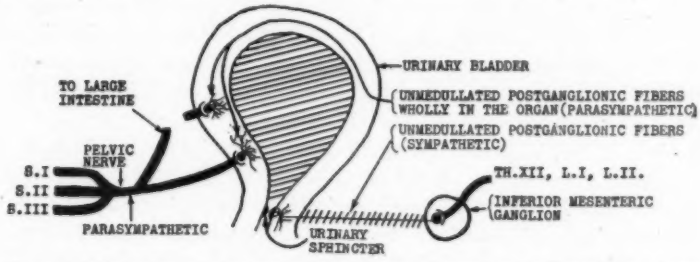

Fig. 7.—Bladder innervature and method of involvement in poliomyelitis. Contents of bladder: The virus plus the toxic products of intestinal absorption have been absorbed and are being excreted. When accumulated in the bladder, absorption by the end-fibers results in local peripheral paralysis of both the sympathetic and parasympathetic systems.

disease is accelerated and its severity is enhanced.[35] Further, when we utilized these factors of virus and enteric toxin, we obtained a fortified antigen which produced a highly potent neutralizing antiserum.[36]

In the light of the facts given, one can easily picture the clinical types of infantile paralysis that are seen in the human being. Certain individuals exist who by their very constitutions have absolutely no symptoms whatsoever even though the poliomyelitis virus may enter their systems.

Another type would be the individual in whom the end-fibers of the gastrointestinal sympathetic and parasympathetic systems might be stimulated by the virus, as a consequence of which the only clinical evidence of disease would be some gastrointestinal irritation or diarrhea.

A third type could have direct extension of the poison along the unmyelinated fibers to the sympathetic ganglion and between the gan-

glionated chains with resulting gut paralysis. At this time, we would find early disturbance of the abdominal reflex arc, a diminution, loss, or lagging to stimulation after repeated stroking.

In a fourth type, the virus merely extends to the somatic nerves and muscular weakness is the result. If this is complete, Landry's paralysis is seen.

In a fifth type, the virus could extend along the sympathetic chain to the point of lessened resistance around the lumbar area and then along the somatic nerve to the intermediolateral area in the cord and secondarily involve the anterior horn cell.

In a sixth type, more severely affected, the virus, after involving the lowermost portion of the sympathetic system, might extend up toward the other localized and unprotected spot, i.e., to the cervical area, and paresis or paralysis of the muscles of the upper extremity would result.

The seventh type would be afflicted with a virus severe enough to bridge the connector gap in the thoracic area and abdominal paralysis would result in addition to the paralysis of the arms and legs.

There is an eighth type in which the poison may be severe enough to bridge the synaptic gap of the end-fibers of the vagus, involve the connector neurone or vagus nerve trunk and simulate bulbar palsy, or in which the virus may travel along the axis cylinders of the vagal fibers to reach and involve the vagal nuclei in the bulb.

A ninth type could exist in which the virus might spread along the sympathetic gray nerves of the vascular network to the brain. Later when the blood stream became filled with the toxic products of intestinal stasis, local reactions would result wherever the virus would be present in the cortex, giving rise to a cortical type of reaction.

A tenth type exists when the virus is absorbed only along the chordae tympani to the nucleus of the seventh nerve.

It would seem to me that only by presupposing an entry by way of the gastrointestinal tract can we explain the symptoms of this disease.

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REACTIONS FROM ALUM TOXOID

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THESE observations are reported to emphasize the age factor in the production of abscesses, and to analyze the immediate reactions (within twenty-four hours) and delayed reactions (protein sensitization) following the use of alum toxoid for diphtheria immunization.

During the fall of 1934 the entire population of 705 children at the Illinois Soldiers' and Sailors' Children's School was Schick tested using controls in each case (Table I). Of these 101 were found positive and were subsequently injected subcutaneously with 1 c.c. of alum-precipitated toxoid at the side of the insertion of the deltoid.

TABLE I

SCHICK STATUS—705 CHILDREN

	POSITIVE	NEGATIVE	TOTAL
Positive protein	1	35	36
Negative protein	100	569	669
Total	101	604	705

All of the children were examined twenty-four hours after being injected for evidence of immediate reactions (Table II). Forty-eight of the 101 had elevations of temperature. Of these, 14 had temperatures of 100° F. or over. Only in one instance did it reach 103° F. Eleven children were admitted to the hospital. This would correspond to remaining home from school in private practice cases. Eight had headache, and two marked swelling of their arms. None vomited.

TABLE II

IMMEDIATE REACTIONS IN 101 CHILDREN TWENTY-FOUR HOURS AFTER ALUM TOXOID INJECTION

	NO.
Temperature above 98.6° F.	48
Temperature 100° F. or over	14
Highest temperature	103°
Admitted to hospital	11
Headache	8
Emesis	0
Marked swelling of arm	2

Abscesses were found in a large percentage from two to three weeks after being injected. There were 22 frank abscesses in the 100 children examined. Three reactions read as indurated areas were followed by scar formation. They presumably formed abscesses later, thus raising

the total number to twenty-five. Some of these opened spontaneously while others were incised. The small closed abscesses were left alone and finally resolved. Table III shows that the abscesses occurred only in children eight years of age and over.

TABLE III

ABSCESSES, SCARS, AND NODULES FOLLOWING ALUM TOXOID

AGE IN YEARS	MALE*	FEMALE*	TOTAL NO.	ABSCESSES	SCARS	NODULES
3	2	0	2			
4	0	1	1			
5	1	2	3			
6	2	2	4			1
7	4	3	7			
8	9	5	14	1	1	
9	5	10	15	6	5	2
10	5	6	11	4	4	1
11	6	6	12	6	3	1
12	4	5	9	1		1
13	4	7	11	5	3	
14	2	4	6			
15	1	2	3			
16	1	1	2	2		
17	0	0	0			
18	1	0	1			
Total	47	54	101	25	16	6

*All white except one colored female twelve years of age.

Of the 25 abscesses, 16 left scars. Of these 15 were examined almost a year after being injected, and in all the scars were still present. The scars were small, varying in size from 2 by 3 mm. to 6 by 15 mm. Their colors ranged from that of normal skin to a faint erythema.

In addition to the scars 3 developed purplish discoloration at the site of the abscesses which persisted for five months.

Palpable subcutaneous nodules were found in 6 of 99 examined five months after their injections. Five of these 6 children were seen again at one year, and only 3 nodules were palpable.

It is interesting to note that while only 1 child had a positive protein reaction before alum toxoid immunization, 14 showed this reaction afterward. Eighty-six of the 101 children injected had received toxin-antitoxin four months to ten years before, the interval being several years in most instances. Records of the other 15 were not available.

The efficacy of a single dose of alum toxoid is shown in this small group. Ninety-eight of 99 children Schick tested in five months' time were found to be negative. The presence of abscesses, including the discharging ones, did not prevent immunization.

COMMENT

The abscesses apparently occurred on the basis of an altered subcutaneous tissue response to an insoluble foreign body in older children. With the same lot of material given to the children both under and over

eight years of age, a larger number of abscesses would have been expected in the entire group if they occurred because of bacterial contamination of the alum toxoid. The following methods were used and should have been sufficient to preclude any contamination during the injections: Syringes and needles were sterilized in boiling water; a separate needle was used for each injection; the caps of the bottles were washed with alcohol before piercing them; and the site of injection was prepared by application of an alcohol sponge and then dried with a piece of sterile gauze. Bacteriologic studies of the abscesses could not be made because of lack of facilities. Abscesses have been noted by Baker and Gill[1] and Kositza.[2] The latter found negative cultures from two incised abscesses and considered the others presumably sterile.

Accordingly, children eight years of age and over should not be injected with alum-precipitated toxoid. However, this agent should be used in the younger groups because of freedom from reaction,[3] efficacy in obtaining a high percentage of Schick-negative reactions,[2-5] and the fact that only one injection is usually required.

The appearance of a positive protein reaction in so many children following alum toxoid is significant and should be studied further.

CONCLUSIONS

1. Abscess formation frequently follows the use of alum toxoid in children eight years of age and over. The abscesses often lead to scar formation.

2. Nodule formation does not occur frequently.

3. Immediate reactions are not serious.

4. A high incidence of protein reactions followed the use of alum toxoid in this series.

5. Alum-precipitated toxoid in single 1 c.c. dose is very effective in establishing immunity to diphtheria.

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HEMATOLOGY AND PATHOLOGY OF LEUCEMIC RETICULOENDOTHELIOSIS

(Monocytic Leucemia) Occurring in an Eighteen-Month-Old Infant

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SINCE the publication of the so-called new or third type of leucemia under the name of "splenocytic leucemia" by Resehad and Schilling-Torgau[40] in 1913, an increasing number of such reports have appeared in the literature. An extensive review of the published cases of monocytic leucemia was undertaken by Merklen and Wolf[34] in 1927, but unfortunately these authors included under this title similar but not true monocytic leucemia. Dameshek,[10] in carefully reviewing the literature on this type of leucemia (1930), was able to isolate but eighteen well-substantiated and fully described cases of monocytic leucemia, including two of this own. Clough[8] (1932) likewise summarized the twenty-two case reports, but Gittins and Hawksley[22] (1933) again reduced the number by eliminating from the list all those cases which, though showing typical hematologic characteristics, were not confirmed by autopsy studies. Additional reports not included in either of the above mentioned reviewers have also been recorded by Böhne and Huismans[5] (1932), Weissenbach[50] (1933), Marchal, Bergeton and Mahoudeau[32] (1933), Labbé, Boulin, and Balmus[28] (1934), Doan and Wiseman[13] (1934), and Sedat[44] (1935).

The great majority of cases of monocytic leucemia have occurred in adults, a fact compatible with the remark once made by Lion and Pernet[31] to the effect that this disorder is an affection of the fifties. Previous to the case reported here, there were but four known instances of this malady occurring in children: first, a five-year-old male child (Rosenthal[41]); second, an eleven-year-old male child (Hannema[24]); third, a thirteen-year-old male child (Lawrence, Josey, and Young[30]); and fourth, an eleven-month-old female infant (Gittins and Hawksley[22]). The case here reported is the fifth in the series.

CASE REPORT

The patient was an eighteen-month-old white male infant with, so far as ascertainable, totally negative family history and normal development. Three weeks prior to hospitalization the child contracted a cold (cough and coryza) simultaneously with a swelling of the lymph gland under the right side of the chin. This was soon followed by progressive enlargement of the other glands of the neck and axillae. Ten days later, and eleven days previous to his entrance

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to the hospital, he was exposed by his sister to measles. Four days after exposure, he was given 10 c.c. of convalescent serum. On the third day following the injection of serum, a maculopapular rash appeared on the lower legs and around the buttocks, which was still present four days later when the patient came into the hospital.

Approximately one week before the child came to our attention, and at about the time the serum was administered, he began to bruise easily, several black and blue spots appearing on various parts of the body and gradually increasing in extent. The mother had noted an occasional bleeding from a crack in his lip, but no bleeding from the nose or gums. About three days after the first appearance of the purpuric spots, a blood count, made by the family physician, registered 2,000,000 erythrocytes, 8,000 leucocytes, and a slight prolongation of bleeding time. No study of stained smears was made at this time. Two days

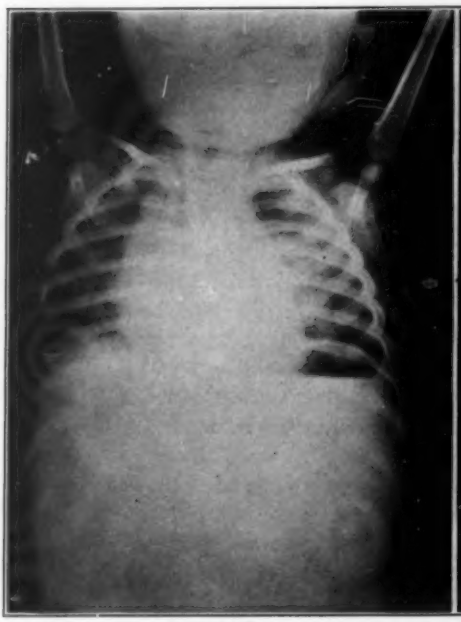

Fig. 1.

Fig. 2.

Fig. 1.—Roentgenogram of chest showing increased width of mediastinal shadow.
Fig. 2.—Roentgenogram of long bones showing slightly increased density of metaphyseal plates.

later the child was given 25 c.c. of the father's blood intramuscularly, followed by a similar amount given just previous to his hospital admission forty-eight hours later.

The physical examination revealed a well-developed, well-nourished male infant to all appearances acutely ill. The skin was warm and moist to the touch, and numerous purpuric spots about 0.5 c.m. in diameter, were noted on the face, back, buttocks, and legs. The conjunctivae of the eyes were moderately injected. The left ear canal contained purulent material and débris and the right some sanguineous discharge. The tympanic membranes were red but not bulging. There was a mucopurulent discharge from the nostrils. Bleeding from the gingival and alveolar margins was evident. The tonsils were moderately enlarged but not inflamed.

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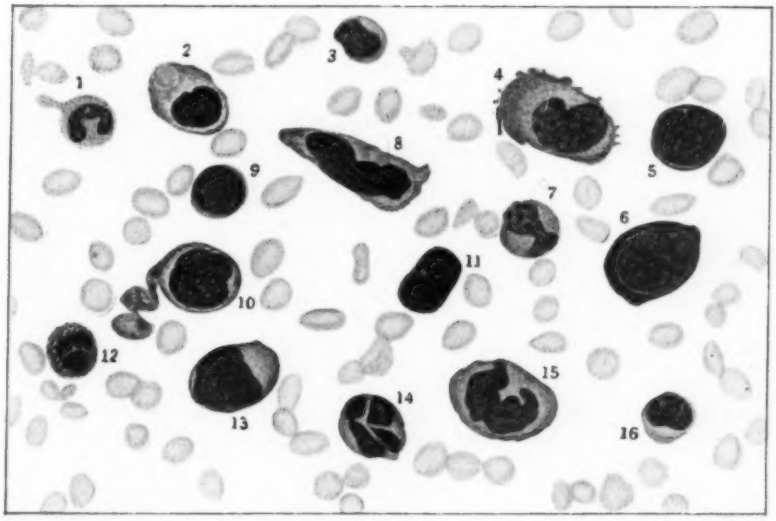

Fig. 3.—Composite drawing of representative hemocytes seen in a smear of peripheral blood, May-Grünwald-Giemsa, camera lucida, oil immersion lens, 10 ocular. *1, 7,* neutrophilic metamyelocytes; *2,* monocyte with an intact erythrocyte ingested in cytoplasm; *3, 16,* mature lymphocytes; *4, 8,* histiocytes; *5,* myeloblast; *6,* lymphoblastic plasma cell (Türk's irritation form); *9,* immature lymphocyte (prolymphocyte); *10, 13,* immature monocytes (promonocytes); *11,* polychromatophilic normoblast in mitosis; *12,* basophilic polymorph; *14,* prolymphocyte with segmented nucleus (Rieder cell); *15,* mature monocyte.

The cervical lymph nodes were increased in size, varying from pea to hazelnut size and presented as palpable, firm masses in all triangles of the neck. The heart, lungs, and genitals were normal, but the abdomen was protuberant with palpable spleen and liver. Both of these organs descended on either side to about 3 cm. below the costal margins. Numerous purpuric spots and petechial hemorrhages were to be seen on the legs, while a large bluish area about 3 cm. in diameter appeared on the buttock over the site of the blood injection. Many maculopapular eruptions were symmetrically distributed over the distal half of the lower legs, being particularly pronounced around the ankles. A generalized lymphadenopathy, the nodules varying in size from 0.5 to 1.0 cm. in diameter and more marked on the right side of the neck than on the left, involved the cervical, postauricular, axillary, inguinal, and epitrochlear nodes.

Owing to the very brief time in which the patient was under hospital observation, the period of blood examination was unfortunately very brief. The total and differential count June 13, 1935, was as follows:

Total erythrocytes	2,500,000
Hemoglobin (Sahli)	36 per cent
Total leucocytes	6,000
Lymphocytes	30 per cent
Monocytes	42 per cent
Neutrophiles	23 per cent
Eosinophiles	0
Basophiles	1 per cent
Plasma cells	4 per cent

The erythrocytes varied considerably in size and shape, with marked achromia. On stained smears, the platelets were much reduced. A few normoblasts were seen and the white cells, as judged by the presence of large numbers of smudge cells (Gumprecht's shadows), exhibited a striking tendency to rapid and easy destruction. The increase in monocytes, many of which displayed an evidence of being histiocytic in type, indicative of a pronounced stimulation of the reticuloendothelial system, was both relative and absolute. (Fig. 3.)

Roentgenographic examination of the chest (Fig. 1) and long bones (Fig. 2) was made on the day of death. In the left lung a slight increase in the markings in the mesial third adjacent to the hilum was noted. The right lung exhibited a similar condition, but with the addition of a soft tissue mass approximately 6 by 2.2 cm. in the right side of the mediastinum. If the aspect of the lung tissue to the right of the mediastinum was produced by glandular enlargement, then the shadow to the left of the mediastinum was probably likewise a consequence of the same pathology, a supposition which was further confirmed by the very noticeable displacement and compression of the trachea approximately 2 cm. to the right. This was almost certainly caused by the enlargement of the glands. In the long bones of the lower extremities the only significant positive finding was the presence of somewhat dense metaphyseal plates with normal epiphyseal lines.

The patient's condition on the third hospital day seemed to be critical, and 200 c.c. of compatible blood from the mother (both the donor and the recipient belonged to Group IV) was given by transfusion into the superficial vein of the neck. At the end of the transfusion the patient developed marked cyanosis. Petechial hemorrhages appeared on both cheeks and the heartbeat became strong and rapid, breathing labored and gasping, and moist râles were heard in both lungs. Administration of adrenalin (2 minims) failed to produce any improvement, and the respiration continued difficult and at increasingly longer intervals until death ensued one hour later without noticeable increase in cyanosis.

AUTOPSY PROTOCOL

Autopsy was performed by Dr. Harold C. Huston within two hours after death and established the following anatomic findings and diagnosis:

Monocytic leucemia; monocytic infiltration of the lymphatic system, enlarged lymph nodes, and subcapsular and submucosal plaques, as well as deeper invasion of various tissues such as thymus, lung, stomach and intestines, liver, pancreas, spleen, epicardium, kidney, and skin; acute pulmonary edema; petechial hemorrhages into skin, subcapsule of kidney, subepicardial and submucosa of the trigon of the bladder; right hydroureter; bilateral interstitial hemorrhage into testes; cervical (right) hematoma after venipuncture (for transfusion).

External Appearance.—The body was that of a white male child about eighteen months old, weighing 32 pounds and measuring 34 inches in length. A 3 by 4 cm. hematoma in the upper right cervical region bore a central puncture mark from which red blood be expressed. A slight amount of blood-tinged, purulent discharge was present in both external auditory canals. An erythematous and papulosquamous rash was noted over both forearms and legs, particularly over the tibial areas. Typical petechial hemorrhages in both antecubital spaces extended to midway of the flexor surfaces. Just above the symphysis pubis was a cutaneous nodule, about 2 by 3 cm. in diameter. A chain of numerous firm and well-defined lymph nodes were palpable in both the anterior and posterior cervical triangles extending down from the mastoid process to the supraclavicular regions on both sides of the neck. Clusters of large, firm nodules were likewise felt in both axillae, the masses being approximately 3 by 3 cm. in size. The epitrochlear nodes were enlarged, the popliteal nodes unpalpable. The inguinal nodes were prominently enlarged on both sides.

Abdominal Cavity.—The stomach and intestines were but moderately distended and the lower abdominal cavity contained about 50 c.c. of clear straw-colored fluid. The liver margin was 6 cm. below the costal edge at the midclavicular line and at the xyphoid process. The diaphragm was at the fifth rib on the right and at the fifth interspace on the left. All the mesenteries showed extensive nodular and cordlike infiltration, especially at the bases and extending outward to the attached gut and diminishing in extent as the process approached the intestine, the last one to two centimeters of the mesentery being but slightly infiltrated, or even normal. The serous surfaces were everywhere smooth and unbroken, except for occasional subserous opaque plaques 1 to 5 cm. in diameter which—upon sectioning—appeared to be an infiltration of grayish white tissue. Nodules 3 to 10 mm. in size encircled the bile duct and followed the great vessels into the pelvis and surrounded the femoral vessels. Clusters of similar but firmer nodules appeared in the inguinal regions. The appendix, approximately 12 cm. in length and 6 mm. in diameter, was retrocecal and of an uncommonly firm consistency. The pancreas was also unusually firm, and its fleshly nodular markings indistinct. The spleen was approximately three times its normal size, its margins rounded, and the capsule smooth. Over the subdiaphragmatic surface of the liver appeared subcapsular opaque mottled areas 5 to 10 mm. in diameter, somewhat confluent and decreasing in extent toward the margins, beyond which they did not extend. Little or no infiltration of the peritoneal surface of the anterior abdominal wall was discernible.

Pleural Cavity.—The thymus was firm and extended from above the sternal notch down to the anterior mediastinum, 10 cm. and 5 cm. respectively, laterally on either side of the middle line, overlapping the lung margin and posteriorly in contact with the great vessels. The lungs filled the pleural cavities almost completely and were but moderately crepitant and firm in consistency. Each pleural cavity contained approximately 50 c.c. of clear fluid. Along the course of the great vessels,

particularly where they passed through the diaphragm, were several firm nodules, ranging in size from 5 to 15 cm. in diameter; similar nodules of firm tissue occurred posteriorly to the thymus.

Pericardial Cavity.—From the pericardial cavity 10 to 15 c.c. of clear fluid was recovered. The heart was unenlarged; the right ventricle was in diastole and the left in systole.

The mouth, pharynx, larynx, trachea, and esophagus were normal.

Thyroid and Thymus.—The thyroid was normal. The thymus weighed 53 gm., or approximately twice that of a normal gland of a child of this age. This tissue was uncommonly firm and on cutting was revealed to be reddish gray in color.

Heart, Aorta, and Other Vessels.—The thickened pericardium was streaked and mottled with grayish opaque areas most noticeable where the sac meets the great vessels and also near the diaphragm. The heart weighed 66 gm. The epicardial surfaces, like the pericardium, were slightly mottled. Multiple punctate subepicardial hemorrhages 1 mm. or less in diameter occurred along the course of the coronary vessels, principally over the left ventricle. No thrombosis of either pulmonary or coronary vessels was noted. The wall of the right ventricle was 2 mm. in thickness; that of the left, 6 to 8 mm. The myocardium appeared normal. The tricuspid orifice was 6.5 cm. in circumference, and the cusps were normal. The mitral ring was 3.5 cm. in circumference, and the cusps were unaffected. The aortic valve was 3.5 cm. in circumference, the leaflets normal, and the aorta itself smooth and elastic throughout its course and nowhere compressed by the periaortic infiltration which extended from just below the diaphragm to the pelvic vessels.

Lungs.—The lungs were but moderately crepitant. Upon compression a frothy thick fluid escaped from the bronchi. Upon sectioning, the lung tissue was found to be unusually wet, yet not hyperemic. The bases were boggy and grayish, and on the left side along the diaphragmatic surface for a distance of 1.5 to 2.0 cm. and into the parenchyma the tissue was fleshy. The right lung weighed 180 gm.; the left, 160 gm.

Peribronchial Glands.—On the right, the peribronchial glands were enlarged and confluent in masses 1 by 3 cm.; on the left they were somewhat smaller. Sectioning revealed the tissue to be fairly firm and reddish gray in color.

Liver and Gallbladder.—The liver, 760 gm. in weight, was slightly rounded at the margin, but the edges of the section were easily approximated; an opaque, reddish gray tissue thickened the capsule over the subdiaphragmatic surface; and along the margins was seen a widely variable, subcapsular, dark red mottling. Upon cutting, the surface was pale and the lobular markings only moderately distinct. The gallbladder, approximately 1.5 by 4.0 cm. in size and with thickened walls, contained thin, watery bile; a firm lymph node appeared at its base, as well as multiple smaller lymph nodes extending in chain formation along the course of the common duct and infiltrating the ampulla of Vater. This was elevated 5 mm. above the duodenal mucosa.

Spleen.—The spleen was left intact with the complete abdominal visceral contents and hence was not weighed, but was estimated to be three times its normal size; its capsule was smooth; the edges were slightly rounded, and, upon cutting, the substance was wet and easily scraped away, revealing the malpighian corpuscles as prominent whitish points. An accessory spleen 1 cm. in diameter was noted. The splenic vessels were surrounded by cords of infiltrated lymph nodes extending to and surrounding the tail of the pancreas.

Pancreas.—The pancreas was found to be extraordinarily firm; the lobular markings were obliterated by subcapsular plaques of tissue extending into the substance of the organ. Upon cutting, the tissue was reddish gray in color with occasional

punctate hemorrhagic areas. The head of the pancreas was embedded in a mass of infiltrated lymph nodes which surrounded the common duct and extended along the bile duct as above described.

Gastrointestinal Tract.—The stomach was moderately distended with gas and undigested curds. The mucosal folds, especially in the cardiac end, were remarkably prominent, and over the mucosa were scattered numerous umbilicated, round, sharply defined elevations 1 to 2 mm. in thickness and 2 to 8 mm. in diameter. These elevations were most numerous in the cardia and absent altogether in the extreme pyloric end. Although the duodenomucosal folds appeared more prominent than normal, no definite nodules were discernible. The ampulla of Vater was elevated 5 mm. above the surrounding mucosa but was not obstructed.

Adrenals.—The adrenals were of normal size, and, upon sectioning, the medullary portion was brownish in color and thickened.

Kidneys.—The right kidney weighed 70 gm.; the left, 65 gm.; in all other respects the kidneys were identical. The perirenal fat was normal. The renal vessels and the ureter were enclosed in a cordlike mass of tissue extending up to and including the pelvis. There was a subcapsular mottling which, when the capsule was stripped away, was apparently due to slightly elevated masses of tissue. After cutting into the kidney, these masses were seen to extend in conelike formation 6 mm. in length into the kidney cortex. Similar but less numerous masses were observed in the medulla of the kidneys. The cortex was 5 mm. in thickness; the medulla, 12 mm. Occasional punctate hemorrhagic areas had occurred in the cortical and subcapsular portions.

Urinary Tract.—Each ureter was contained in a cord of tumor tissue, reaching from the kidneys to the bladder. Neither ureter was occluded, but the one on the right was slightly dilated from the level of the pelvic rim to the renal pelvis. Throughout both ureters were noted pinpoint elevations of the mucosa beginning in the kidney pelvis and diminishing in frequency toward the bladder, which contained approximately 25 c.c. of slightly turbid urine. Except for occasional submucosal hemorrhages over the area of the trigon, the wall and mucosa were normal.

Generative Organs.—The testes were of equal size. Upon sectioning, the tissue was soon to be deep red and bulged markedly above the cut surface. The prostate and the seminal vesicles were normal.

Lymph Nodes in General.—Dense infiltration occurred in the following nodes: the anterior and posterior cervical nodes, the subclavicular, the inguinal and epitrochlear, the anterior mediastinal and peribronchial, the periaortic (especially from just above the diaphragm and including the celiac axis), and the intercalated nodes and the mesenteric circulation. Among the infiltrated vessels were noted: the splenic, the duodenal, the renal arteries, and the vessels extending into the pelvis and into the inguinal nodes and the femoral triangle.

Muscular and skeletal systems: Grossly normal.

Brain, meninges and spinal cord: Not examined.

Postmortem Bacteriology.—The smear made from materials obtained by lung puncture revealed no bacteria, but upon culture the following organisms were identified: (1) *Micrococcus luteus;* (2) *Streptococcus anhemolyticus;* (3) *Streptococcus alphahemolyticus;* (4) a large gram-negative diplococcus with the cultural requirements of *B. influenza.*

HISTOLOGIC FINDINGS

The histologic examination was made on sections from tissues fixed in 10 per cent formol-Zenker, embedded in celloidin and stained with hematoxylin and eosin.

Lungs.—The section disclosed extensive noninflammatory edema. The blood vessels contained many mononuclear cells having abundant cytoplasm. There was

considerable perivascular infiltration with similar cells, the nuclei of which were often kidney-shaped. There was likewise an accumulation of these monocytic cells in tissue areas independent of blood vessels. Only a few lymphocytes and polymorphonuclear cells were seen.

Lymph Nodes.—These nodes were almost entirely replaced by large mononuclear cells which were seen to infiltrate the adjacent tissues as well; the normal gland architecture was destroyed, and only a few remaining foci of lymphoid cells were left intact. The nuclei of the mononuclear cells varied greatly in configuration; occasional mitotic figures were seen but were not numerous (Fig. 4).

Liver.—This organ showed great periportal infiltration both with large mononuclear and with a lymphoid type of cells. A slight accumulation of these cells was also found in the sinusoids (Fig. 5).

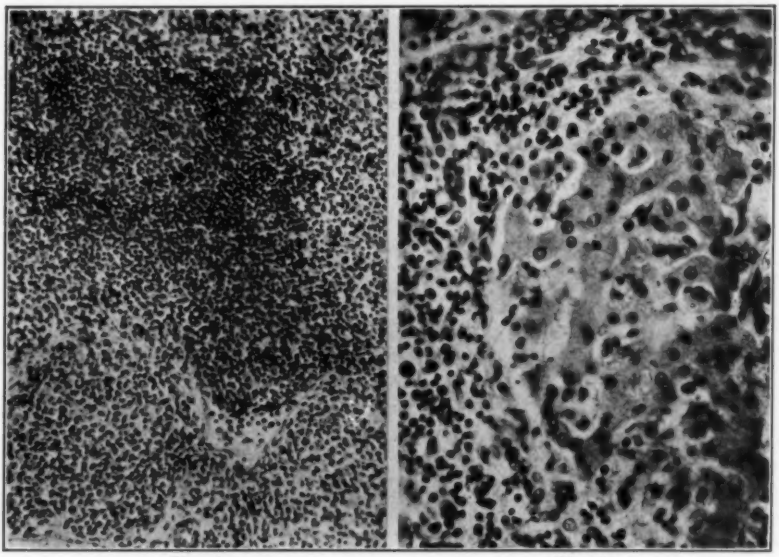

Fig. 4. Fig. 5.

Fig. 4.—Photomicrograph of a section of lymph node showing a dense mononuclear infiltration around the lymphoid follicle. Hematoxylin eosin, ×240.

Fig. 5.—Photomicrograph of a section of liver, showing an island of hepatic parenchyma surrounded by a proliferating mononuclear cell mass. Hematoxylin eosin, ×535.

Kidney.—The section revealed areas of infiltration with mononuclear cells, chiefly about the blood vessels, in the pelvic connective tissue and—to a much lesser degree—in and about the glomeruli. Intermingled with these large mononuclear cells were occasional lymphoid foamy cells.

Heart.—The epicardial fat was infiltrated with large mononuclear cells; a number of vessels within the myocardium were also thus infiltrated, but to a lesser extent.

Thymus.—The entire gland revealed such an enormous infiltration of mononuclear cells that little recognizable thymus tissue was left; many of these cells suggested immature marrow elements, but no erythropoiesis here or in any of the other organs was indicated.

Pancreas.—The interstitial tissues were invaded by monocytic cells to the extent that approximately half of the tissue in the sections examined was thus infiltrated. Even the islets appeared to be partially replaced by this type of tissue, though many normal ones remained.

Bone Marrow.—The marrow of the rib was completely replaced by the same identical cells found infiltrating the numerous other organs. Erythropoiesis was altogether lacking. Eosinophiles and megakaryocytes were but occasionally seen. There was a pronounced absorption of the bone. The marrow of the vertebrae presented similar findings, but with a lesser extent of bone absorption (Figs. 8 and 9).

Testes.—There was extensive, diffuse interstitial hemorrhage. Much cellular infiltration was seen in the epididymus, but none in the testicles.

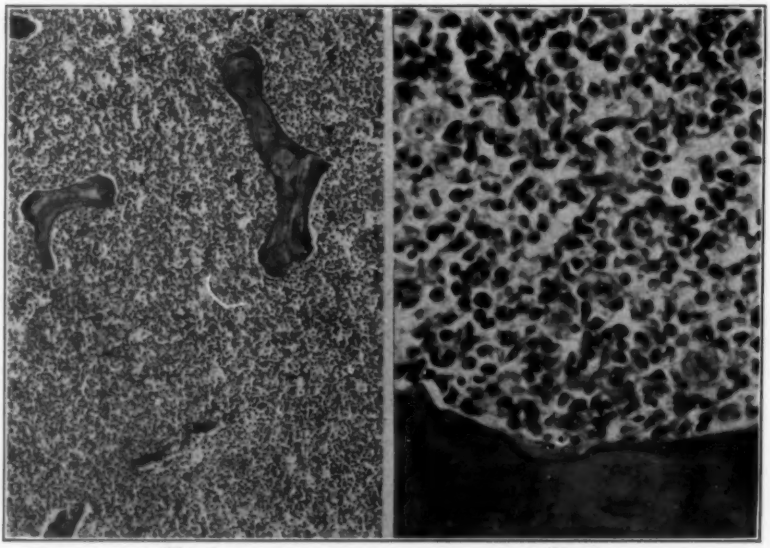

Fig. 6. Fig. 7.

Fig. 6.—Photomicrograph of bone marrow (rib) showing complete infiltration of marrow spaces with mononuclear cells. Only a few immature megakaryocytes (megakaryoblasts) are visible. Hematoxylin eosin, ×125.

Fig. 7.—Photomicrograph of a portion of the rib marrow shown in Fig. 6. Hematoxylin eosin, ×600.

Stomach.—Cellular infiltration was extensive in the mucosa and more extensive in the submucosa, where masses were formed resembling tumors but composed of the same histologic elements as the infiltration elsewhere.

Adrenal.—This section was free from infiltration although adjacent tissue revealed a certain amount of monocytic invasion.

Skin Nodule.—The nodule was composed of infiltration of the subcutaneous tissues with mononuclear cells associated with small areas of hemorrhage.

Subpleural Nodules.—These nodules demonstrated infiltration of the pleura and the underlying adipose tissue with the mononuclear cells.

Paravertebral Nodule.—This consisted of a tumorlike growth of the cells and of fragments of hard bone and degenerating muscle.

Spleen.—The architecture was well preserved, there being relatively little evidence of the infiltrative process that involved the other lymphoid organs. The reaction centers of the malpighian corpuscles were large, and in the pulp a few large mononuclear cells were seen, together with a moderate number of eosinophiles, myelocytes and an occasional megakaryocyte.

COMMENT

With its typical blood picture and its characteristic changes in the various organs which abounded in histiocytic elements, the proliferative disease of the reticuloendothelial system appears to be a distinct clinical, hematologic, and pathologic entity. Clinically this condition is indistinguishable from any other type of acute leucemia, their com-

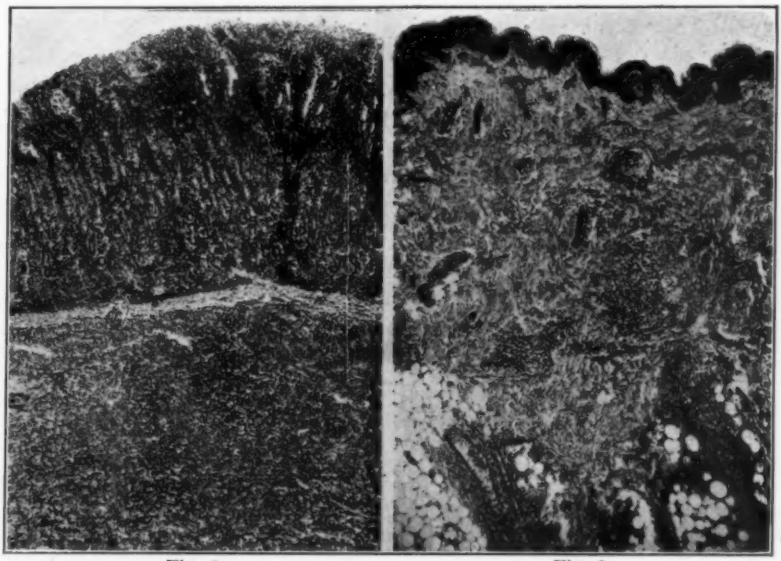

Fig. 8. Fig. 9.

Fig. 8.—Photomicrograph of the wall of stomach showing mononuclear cell infiltration in the mucosa and submucosa. Hematoxylin eosin, ×65.

Fig. 9.—Photomicrograph of skin nodule showing mononuclear cell infiltration in the subcutaneous tissue. Hematoxylin eosin, ×60.

mon symptoms being an acute course, anemia and pallor, hemorrhagic tendency, and hyperplasia of the hemopoietic organs. Occurrence of this type of leucemic state in an infant demonstrates that age cannot be said to be an exclusive etiologic factor. The exact nature of the stimulus activating this multiple hyperplasia is extremely difficult to ascertain. Sepsis of infection has been suggested as the causative factor in many cases of reticuloendotheliosis recorded in the literature, but in the present case it scarcely seems plausible that a cold, or even a cold possibly complicated by measles (if such might have been the

nature of the rash, rather than that of serum reaction), would produce an infection or sepsis overwhelming enough to constitute the sole predisposing factor responsible for so remarkable and so extensive a change in the organs. It would seem more reasonable to think that the lymph gland condition was already established prior to the onset of the upper respiratory infection, but to such a mild degree as to have escaped the attention until stimulated by the infection to a noticeably acute activity. The pathologic picture of the organs suggests a similarity to multiple neoplastic or malignant hyperplasia of the histiocytic apparatus. Because the proliferative alterations were so generalized and so extensive, the malady was inevitably fatal. No therapeutic measures are successfully applicable to such a widely and rapidly spreading process.

Perhaps the most difficult aspect of this disease is a proper interpretation of the blood picture. In spite of the extreme hyperplasia of the reticuloendothelial system, the peripheral blood reflects no increase in white blood cells, the one feature which stands out in sharp contrast to the conspicuous leucocytosis produced in the other reported instances of monocytic leucemia.

In typical monocytic leucemia, as ordinarily encountered, the peripheral blood commonly shows, as a consequence of the great increase in monocytes, a high degree of leucocytosis. In the case here reported no such increase in the total count of the white blood cells occurred; in fact, and to the contrary, the actual figure indicates a slight leucopenia. The stained smears revealed a considerable number of atypical monocytes (which some observers may interpret as being identical with histiocytes). It must be emphasized, however, that these atypical monocytes or histiocytes seen in the peripheral blood to the extent of 42 per cent of the total cell count are the pathologic cells upon which the diagnosis is based. If this increase in pathologic cells is determined upon as the diagnostic criterion, then the condition may be termed "leucemic reticuloendotheliosis." It is in the section of the capillaries of the various organs, but most conspicuously in the lung capillaries, that the true leucemic nature of the blood is revealed. Congested in practically all small vessels of the lungs are found an exceedingly great number of mononuclear cells identical with the pathologic cells seen in the peripheral blood. This may be interpreted to mean that the leucemic cells were in some manner impacted in the lung capillaries in such great aggregations as to allow relatively few free cells to escape into the peripheral circulation. This phenomenon must always be considered in any case of acute leucemia in which the peripheral blood fails to indicate proliferation of the pathologic cells. Also probable is the supposition that of the comparatively few cells released into the systemic circulation many may be relodged in the

capillary beds of various other organs where they may set up proliferation, producing the foci of monocytoid metaplasia. It may thus be seen that the total leucocyte count, or a merely quantitative alteration in the peripheral blood, should therefore never be relied upon for an infallible diagnosis of leucemia; and for this reason the term "aleucemic leucemia" should be discarded. The most important and reliable evidence in the diagnosis of leucemic states is the qualitative changes in the white cells themselves, particularly as to immaturity and relative increase. Those cases of proliferative diseases of the hemopoietic organs, such as lymphoblastoma and myeloma, in which there are neither significant, quantitative or qualitative changes of the leucocytes, are not to be labeled "leucemic," even though the pathologic changes are identical with those seen in true leucemias. Promiscuous application by some authors of the term "aleucemic leucemia" to these proliferative diseases with practically normal blood pictures has led to numerous erroneous conceptions of leucemic states.

The pathologic cells involved in this case are large mononuclear cells, some of which most certainly are the typical monocytes seen in the normal blood, while others are extremely atypical and abnormal. Averaging from 25 to 50 microns in dimension, these cells are usually slightly oblong or oval in shape and contain a relatively large amount of cytoplasm, rather flexible in appearance, and filled with somewhat granular basophilic protoplasmic substance stippled with a varying number of very fine azurophilic granules; the nucleus is either rounded or irregular in outline and often most fantastic in configuration. The chromatin material is mostly finely granular and, in some instances, even appears quite reticular; in many of the cells a few nucleoli are visible. The general nature of these cells suggests at once their immaturity, and they may be regarded as representing various maturation stages of the monocytic series, as depicted in Section III in the monophyletic scheme published earlier by the author. Hence the qualitative blood picture is in complete conformity with that encountered in typical monocytic leucemia with quantitative increase in the total cell count. It therefore reasonably follows that "monocytic leucemia" is a synonym for leucemic reticuloendotheliosis, the former term being a clinical nomenclature and the latter a pathologic concept.

The case here reported likewise suggests the possibility of its serving as a guide for the definition of the reticuloendothelial system. The organs which, in this instance, showed the greatest degree of histiocytic proliferation were the bone marrow, lymph nodes, the entire gastrointestinal tract, and the thymus. In all these organs the normal histologic architecture was effaced because of pronounced infiltration of these tissues by the pathologic cells. The submucosa of the whole gastrointestinal tract is the seat of marked monocytic proliferation.

Next in order of hyperplasia were the lungs, liver, kidneys, pancreas, subcutaneous tissues, the adipose tissues surrounding the heart, the epididymus, and the serous membranes. In the skin the infiltration of the histiocytic elements often presented nodular appearance. Organs displaying a relatively small amount of histiocytic infiltration were the adrenals, heart, and spleen. It is worthy of note that the spleen, the organ most commonly regarded as the reticuloendothelial organ *par excellence*, was comparatively uninvolved in the extensive hyperplastic changes seen in nearly all other organs. Although a seat of extensive hemorrhages, the testicles evinced no histiocytic proliferation; but the epididymus, on the other hand, displayed a striking infiltration. The adrenal glands, as judged from experimental studies employing vital staining, usually contain a fair amount of reticuloendothelial elements; but in this case the adrenals revealed but relatively slight involvement. Unfortunately the central nervous system was not examined, but it is quite conceivable to suppose that the perivascular tissue around the capillaries—particularly the hypophysis—may have been infiltrated with the pathologic cells.

Study of the hematology and pathology of a case such as here reported leads to the consideration of the nosologic ventures attempted by a few observers. Epstein[14] suggests the group term of "histiocytomatosis," of which he recognizes the following four subgroups: (1) storage: Gaucher's disease, Niemann-Pick's disease, xanthomatosis; (2) infectious proliferative (granulomas): Hodgkin's disease; (3) hyperplastic: aleucemic and leucemic; and (4) dysplastic: reticuloma, endothelioma. Bock and Wiede[4] classify the diseases of the reticuloendothelial system under two general groups of histiopathic and hemopathetic reticuloendotheliosis; their conception is logical and to some degree influenced the classification here proposed.

Classification of Reticuloendothelioses

I. Hemopathetic (leucemic) reticuloendotheliosis (reticuloendothelial system as a hemopoietic organ with a definitely pathologic change in the circulating blood):
   (1) Monocytic leucemia (acute and chronic)

II. Histiopathic (nonleucemic) reticuloendotheliosis (reticuloendothelial system as a metabolic organ with no blood pathology):
   (1) Storage diseases:
      (a) Kerasinosis (Gaucher's disease)
      (b) Lecithinosis (Niemann-Pick's disease)
      (c) Steatosis or cholesterosis (Schüller-Christian's disease)
      (d) Xanthomatosis (local and general)
      (e) Amaurotic family idiocy (Tay-Sachs' disease)

(f) Hemochromatosis
(g) Diabetic lipemia

III. Infective proliferative reticuloendotheliosis (reticuloendothelial system as an organ of defense with abnormal but nonleucemic blood picture):
(1) Specific infections, particularly typhoid fever.
(2) The so-called "aleucemic" reticuloendotheliosis (Letterer-Siwe)

IV. Dysplastic malignant reticuloendotheliosis (reticuloendothelial system as a seat of neoplastic process with no significant blood changes):
(1) Lymphogranulomatosis (Hodgkin's disease)
(2) Reticuloma and endothelioma.

It must be explained that the above classification is based primarily upon the most fundamental points of distinction among various types of reticuloendothelioses and therefore is not necessarily mutually exclusive. In certain specific instances more than one fundamental characteristic may coexist, but the nosologic position of each type may be defined with a fair degree of clearness by this suggested classification. Furthermore, it must be admitted that the majority of the diseases involving proliferative change in the histiocytic system are obscure as to etiology, save in specific infections as recently summarized by van Creveld and Ter Poorten;[49] consequently the above classification cannot but be tentative and must be amended as our knowledge of such diseases increases. Nevertheless, it must be emphasized that there exist distinct varieties of clinical and pathologic conditions characterized by proliferative and irreversible changes in the histiocytic apparatus which must be grouped together under the general caption of "reticuloendothelioses," or the diseases of the reticuloendothelial system, a chapter as yet unwritten in medicine.

SUMMARY AND CONCLUSIONS

1. A case of reticuloendotheliosis (monocytic leucemia) in an eighteen-month-old male infant with clinical, hematologic, and pathologic findings is reported.

2. This disease constitutes a clinical and pathologic entity that may occur at any age period. The case here reported is next to the youngest patient so far recorded in the literature.

3. A tentative classification of the diseases of the reticuloendothelial system is suggested although, in view of the inadequate knowledge of these diseases at present available, such an attempt may be regarded premature. This group of diseases, however, constitutes a distinct chapter as yet unwritten in any system of medicine.

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RATBITE FEVER

Report of a Case With Serologic Observations

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RATBITE fever is a disease endemic over the greater part of the world and yet rare enough to be somewhat of a curiosity. Its clinical importance is minimized by reason of its infrequency, while the typical diagnostic history and the availability of therapy specific in treatment assure a favorable prognosis.

Considerable attention has been paid to the disease in Japan, where it is known as sodoku, and where the causative organism was isolated and described.[1] Wilcox[2] recognized the first case in this country as early as 1840, and a recent tabulation has shown seventy-five fairly authentic cases in the American literature.[3] We are reporting another case chiefly because of the excellent opportunity it has afforded for studying the serologic changes.

The organism first described by Futaki and his coworkers as the cause of ratbite fever was named by them *Spirocheta morsus muris*. Later workers have considered the name to be inappropriate and have tended to place it in the genus Spirillum. It has also been the popular opinion that the organism is identical with the older species *Spirillum minus*, and most authoritative texts now apply this name to both the mouse and true ratbite strains.

A good review and general discussion of the disease may be found in Bayne-Jones' monograph,[3] while the work of Solomon and his associates[4] provides an excellent clinical study.

Wassermann tests were recorded eighteen times in the series reviewed by Bayne-Jones. In three of these the reaction was positive, but concurrent syphilitic infection seemed probable in two and possible in the third. From these data it was concluded that the Wassermann reaction is negative in ratbite fever when syphilis can be ruled out. Standard textbooks subscribe to the same belief.

In 1934 Edelman and Haber[5] reported three cases of ratbite fever in which the serologic reactions were positive. In each of these instances the precipitin test (Kahn) was strongly positive, while the complement fixation reaction was changed but slightly. These workers were unfortunate in not being able to recover the spirilla from their cases although the histories were conclusive of the correct diagnoses.

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We have been able to recover the typical organism from our patient and have also been fortunate in seeing him early enough to obtain a *primary negative serologic report.*

CASE REPORT

D. M., a six-and-one-half-year-old son of Italian parents, was first seen in the out-patient department because of enlargement of the inguinal glands and possible infection of the right great toe. The patient had always been well and except for the usual exanthemas his past history was negative. The family history was irrelevant.

Three weeks prior to the out-patient visit a rat had bitten the child while he was in bed. There was only a slight amount of bleeding, and hot "soaks" were applied. Healing was rapid, and no further symptoms were noted until seven days after the incident when the glands in the right groin became swollen and the patient

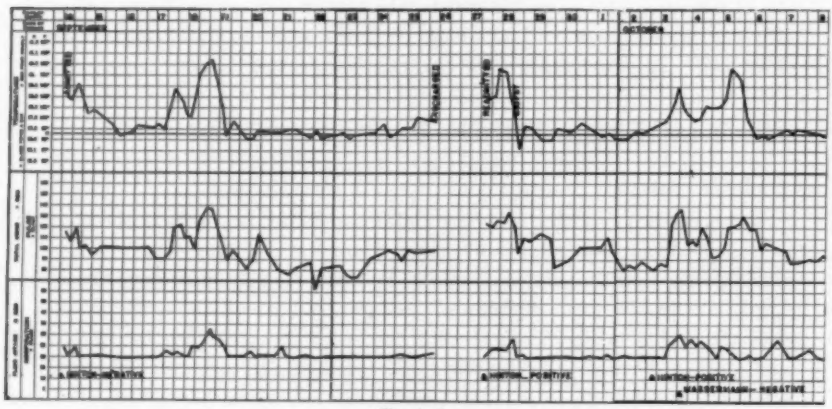

Chart 1.

NOTE.—Out-patient visits following discharge provide the following data: November 13, 0.45 gm. of neoarsphenamine given; Hinton test doubtful.

was feverish. Ice bags were applied locally, and the glands diminished in size. This episode was followed by ten days of normal health before the glands again became enlarged and the great toe red and painful. The temperature was 101° F., and no other symptoms were noted.

Physical examination in the out-patient clinic revealed a well-developed and well-nourished child who was alert, cooperative, and not acutely ill. The temperature was 102° F., the pulse rate 115, and the respirations normal. Except for congenital absence of the right pectoralis major, the positive findings were limited to slight swelling and discoloration of the right great toe with accompanying enlargement and tenderness of the regional glands. There were also a few shotty nontender nodes palpable in the left groin. There was no rash, no evident lymphangitis, and no generalized adenopathy. The patient was admitted to the hospital with diagnoses of cellulitis of the right great toe, inguinal adenitis, and possible ratbite fever.

Course.—Since the history suggested ratbite fever, a venipuncture was done immediately and 3 c.c. of blood were injected subcutaneously into a guinea pig. Blood for culture and Hinton test was obtained at the same time and both were negative.

During the first forty-eight hours the child's temperature gradually fell from 103° F. to normal and remained there for two days. This was followed by a three-day period of fever with the temperature ranging between 100 and 105° F., although the patient felt and acted quite normal. Blood was taken at the height of this fever for inoculation into guinea pigs. The temperature then remained near normal for almost a week, and it was deemed advisable to discharge the child since the adenitis had also subsided and spirilla had not been demonstrated in the animals.

The day following discharge he was again seen in the out-patient clinic with a temperature of 102° F. and an exacerbation of the local adenopathy. He was readmitted and blood was again taken for animal inoculation and for serologic tests. His temperature rose to 105° F. the next morning, and, since there were no other symptoms, it was considered safe to remove one of the large superficial glands. Biopsy was done, and two nodes, each measuring about 2 cm. in length, were obtained. One of these was sent to the pathology laboratory,* and the other was emulsified, examined by dark-field; 3 c.c. of the emulsion were injected into the peritoneal cavity of a guinea pig. Following the biopsy, the patient's convalescence was

Chart 1—Cont'd.

November 27, 0.6 gm. of neoarsphenamine given; Hinton test negative.
December 11, 0.45 gm. of neoarsphenamine given; Hinton test negative.

entirely satisfactory, the temperature remaining normal for five days. The wound healed cleanly, and the sutures were removed on the seventh day.

The Hinton test on the blood taken at the time of readmission was now reported as positive, as was the test on a sample taken four days later. There were two more recrudescences of fever during the next two weeks, and, on the seventeenth day following operation, spirilla were demonstrated in the blood of the guinea pig inoculated with the glandular emulsion. Treatment with neoarsphenamine was begun immediately after the confirmation of the diagnosis, and the schedule of dosage has been included in the accompanying chart. A rash was noted during only one of the periods of hyperpyrexia. It took the form of a rather vague blush on the abdomen and chest and was neither eruptive nor distinctive. The white cell count varied between 12,000 and 25,000 leucocytes per cubic centimeter throughout the illness, with the peak of polymorphonuclear increase coinciding with the peak of temperature.

*Examination of the tissue prepared by both ordinary and silver impregnation methods did not demonstrate the organisms. Considerable hyperplasia and chronic inflammation were present.

DISCUSSION AND CONCLUSION

The spirilla in this instance were recovered from a guinea pig inoculated with an emulsion of a gland obtained at biopsy. The organisms were later also recovered from several of the animals into which the patient's blood had been injected. Unfortunately the latter animals had been kept in the same cage with the known infected one. Further work is now in progress to determine whether the organism was actually recovered from the blood stream or merely that contact contagion is possible among guinea pigs.

The organism was apparently morphologically typical of the species described by Futaki. They were easily found in the blood of infected animals both by direct smear and by examination with the dark-field. They varied in length from 2 μ to about 6 μ, while the spirals varied from 3 in the shorter forms to 5 in the longer. The rapidness of their movement as observed in fresh preparations was most striking and would seem to render confusion with other possible blood stream invaders improbable.

A definite change in serologic reaction took place in this case between the twenty-first and thirty-fifth days after primary infection. Several repetitions of the Hinton test were positive during the active phase of the disease and consistent negative results were obtained only after five injections (1.1 gram total) of neoarsphenamine. It will be seen that one specimen taken during a decline of the fever was negative, and it is possible that pyrexia may tend to influence temporarily the serologic state. The Wassermann test was made twice and was negative even when the Hinton test on one of the same specimens was positive. The serologic reaction by the Kahn method was not investigated.

Syphilis in this case can be definitely ruled out by (1) the initial negative Hinton test, (2) an entirely unsuggestive personal and family history, and (3) the negative serologic reports on samples of both maternal and paternal blood. Our observations consequently confirm those of Edelman and Haber and show conclusively that the Hinton test is positive during the active course of ratbite fever. This should prove of interest and value in governing the extent of therapy in the disease and also of importance in syphilitic patients receiving fever treatment through the medium of this spirillum.

We wish to express our appreciation to Dr. A. Watson Sellards, of the Department of Tropical Medicine, for his assistance in the isolation and identification of the organism. We are also indebted to Dr. P. J. Mahoney, who performed the biopsy and supervised the postoperative care of the patient.

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ANAPHYLACTOID OR ALLERGIC PURPURA

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THE word "purpura" merely signifies a cutaneous hemorrhage. The classification of purpuras is still quite confusing. The types to which I have reference as allergic are those cases in which the hemorrhages appear to have a definite relationship to the ingestion of certain foods or drugs.

The hemorrhagic lesions may vary in size from small petechiae to large ecchymoses. They may vary in number from two or three lesions to many lesions covering the entire body. The apparent sites of predilection for these hemorrhages appear to be the extremities, particularly the lower extremities. The children observed in the following cases reported have been observed very closely in order to rule out other possible causes for the lesions than those mentioned above.

The tendency has been to include in the allergic type of purpura only those cases which have been known as the Schönlein-Henoch type. These types, as is generally known, are accompanied by joint and intestinal symptoms. There are also cases which have been classified as purpura simplex and idiopathic purpura, involving only the skin without joint or abdominal symptoms, many of which cases I feel may have an allergic basis.

Most of the cases of allergic purpura occur particularly in children and young adults. They may be accompanied by other lesions of the erythema group of diseases, one reason for considering sensitization as a possible cause for these lesions. Many of these cases also appear to be seasonal occurring especially during the spring and summer months.

A brief review of the literature pertaining particularly to purpura of this type only is of interest. Osler[1] reports that Wilan in 1808 first described a case of purpura associated with violent vomiting, excruciating pains in the bowels, and anasarcous swelling of the legs, thighs, and hands. Henoch[2] in 1868 described a similar case with abdominal symptoms, and since that time this particular type of case has been referred to as Henoch's purpura. Osler[3] later called attention to three of these patients who had been operated upon because of abdominal colic and states that the greatest care should be taken first to get a full history which might bring out the facts of previous attacks either

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of skin lesions, of arthritis, or of abdominal crises and second to make the most careful inspection of the skin for angioneurotic edema, purpura, or erythema.

At about this time Burrows[4] described a patient who was operated upon because of severe abdominal pain. The next day on inspection of the body, petechiae were noted. This case is of interest because a similar attack occurred a month later following the ingestion of chocolate. This is one of the first cases I have been able to find in which a food relationship to the symptoms had been noted. Fraser[5] reports two similar patients who were operated upon. Gracie[6] also described such patients who were operated upon. The abdominal pain in these cases is due to infiltration of the intestinal wall with blood and serum.

Withington[7] called attention to four skin affections which are met with more or less commonly and which seem to have a close relationship with one another. These were (1) erythema multiforme and nodosum, (2) urticaria, (3) angioneurotic edema, and (4) purpura. Osler[8] later also described, in addition to the above, a necrotic type of lesion in which an ulcer is left following a localized infiltration of blood and serum. In 1914 Osler[8] drew attention to the possibility of some forms of purpura possibly being anaphylactoid phenomena in persons sensitized to certain substances including foods. At the same time he thought that the malignant purpura of the specific fevers might be anaphylactic phenomena.

Similar cases have been described by Landsberger,[9] Alexander and Eyermann,[10, 11, 12] Barthelme,[13] Kahn,[14] and others. Alexander and Eyermann drew particular attention to food as an etiologic factor in the cases which they reported. They attempt to ascribe the cause of the purpura in their cases in which no edema surrounded the hemorrhagic areas as due either to a passage of red blood cells without serum elements, which is most unlikely, or an actual capillary rupture and therefore an atypical lesion of allergy. Goldstein[15] thought these cases were characterized by morphologic and functional changes in the capillary mechanism resulting in abnormalities in the structure of the capillaries, in current pressure and filling abnormalities and variations in the permeability of the capillary endothelium. He does not think it is essentially a hemorrhagic disease and has no characteristic findings.

Cases of purpura occurring after the ingestion of drugs have been reported by Peshkin and Miller,[16] Bickford and Tilghman,[17] and Loewy.[18] Purpura occurring in these cases may be either of the thrombocytopenic or nonthrombocytopenic type. Peshkin and Miller's case presented a thrombocytopenia after quinine with positive skin and passive transfer reactions to quinine. Bickford and Tilghman's cases occurred following the use of arsphenamine. Loewy's cases due to

the use of sedormid were thought due to an allergotoxic effect in which a peculiar susceptibility due to gradual sensitization of the patient must be assumed.

Bradburn,[19] H. Coke,[20] and Kugelmass[21] all have reported cases of Henoch's purpura which have been associated with infection. Symptoms disappeared with removal of the foci.

The following cases occurred in children between the ages of three and twelve years. A positive allergic history was present in most of the patients, as will be noted. In several of these patients purpura was noted during the routine examination of the child, and on later observation a definite association between the purpura and the ingestion of specific foods was noted.

The following cases are those which might be classified as the Henoch type of allergic purpura.

CASE 1.—B. R., female, Dr. J. Rossen's patient whom I saw with him on several occasions. At five and one-half years of age, the child had swelling of right ankle and right knee followed by swelling of left ankle and knee also. There was abdominal pain and fever of 100.4° F. for two days. She complained frequently of "growing pains." The tonsils and adenoids were removed at seven years of age because of continued rheumatic symptoms. At eight years of age she continued to have pains in her joints and abdomen and at that time had dermatitis with papules and vesicles as well as fissures on the legs, hands, and feet. She was referred to a dermatologist who made a diagnosis of dermatitis herpetiformis. At ten years of age she was seen with swelling of her right ankle and purpuric spots over the extremities, accompanied by abdominal pain. There were some necrotic areas in some of the purpuric lesions. Accompanying the above symptoms was a temperature up to 103° F., chills, bloody stools, and diarrhea.

Blood examination at this time showed R.B.C., 4,340,000; lymphocytes, 50 per cent; eosinophiles, 8 per cent; polymorphonuclears, 41 per cent; and basophiles, 1 per cent.

Sepsis was suspected, and an unfavorable prognosis was given by a consultant. The child was put on broths only, and all of her symptoms cleared up promptly. It was at this time that Henoch's type of purpura was suspected, and by an elimination diet milk was found to be the causative factor. A scratch test for cow's milk gave a positive reaction.

Several months later the child entered the Jewish Hospital where a mastoid operation was performed. She developed a temperature elevation following the operation, also purpura and joint symptoms again. Milk was withdrawn from her diet and all symptoms cleared up.

Five years later she was seen at St. Mary's Hospital with a similar attack.

Blood examination at this time showed R.B.C., 2,900,000; W.B.C., 14,200; eosinophiles, 8 per cent; basophiles, 0; young forms, 0; myelocytes, 2 per cent; stab cells, 10 per cent; segmenteds, 68 per cent; lymphocytes, 16 per cent; and monocytes, 4 per cent.

A history at this time recorded the taking of evaporated milk for three months before entry into the hospital. The onset of symptoms was associated with the ingestion of milk. X-ray films of joints and blood cultures at this time were negative. Milk was discontinued again, and all the symptoms disappeared except

a few necrotic skin lesions on her feet, which were diagnosed by a dermatologist as papular necrotic tuberculides. There was no positive allergic history in this child, but there was a history of a serum reaction following the injection of diphtheria antitoxin for a faucial diphtheria at four years of age.

Unfortunately we have not seen this child for the past two years. The sensitivity to milk was quite striking in this particular case.

CASE 2.—F. K., aged four years, female, was first seen in October, 1933, with large ecchymotic areas on the arms and buttocks which had been present for several days. The physical examination was negative except for the purpura.

Blood examination showed R.B.C., 4,590,000; W.B.C., 10,900; hemoglobin, 65 per cent; segmenteds, 28 per cent; stab cells, 0; lymphocytes, 70 per cent; eosinophiles, 1 per cent; and monocytes, 1 per cent.

Coagulation time of venous blood was 5 minutes; the clot retraction time was 1 hour; the bleeding time was less than one minute.

Diagnosis was made at that time of symptomatic purpura.

On Sept. 22, 1934, one year later, she again entered the clinic with purpuric areas on the body, legs, and buttocks which had been present for twenty-four hours. Blood examination at this time showed W.B.C., 8,900; R.B.C., 4,500,000; hemoglobin, 90 per cent; platelet count, 280,000; and bleeding time, 1 minute.

On Oct. 10, 1934, one month later, the child was seen having a typical asthmatic attack. Accompanying this was some abdominal pain and purpura. After a careful dietary history and diary for possible offending foods, tomatoes were removed from the child's diet, and she remained free of purpura for a week. The following week tomatoes were fed again, and purpura reappeared with some abdominal pain but no diarrhea. When she returned to the clinic several months later, the mother stated that the child had not eaten tomatoes and that purpura had reappeared. On checking diet through a diary again, and by means of elimination, chocolate was noted as the cause of the purpura accompanied by abdominal pain at this time.

This case demonstrated the diagnosis of an allergic purpura being made before there was evidence of any other allergic manifestation in the child or antecedents previous to the illness noted above.

The following cases are those in which purpura alone was noted, without joint or abdominal manifestations.

CASE 3.—R. F., male, three years old, entered clinic with complaint of asthma and hives. He was observed at intervals over a period of a year with repeated asthmatic attacks. From time to time during this period, purpuric lesions varying in size from ⅛ to ½ inch in diameter were noted on the upper and lower extremities. A diet diary was kept, and pop corn was noted as the offending food. The ingestion of pop corn on two subsequent occasions produced the purpura unaccompanied by hives or asthma.

CASE 4.—B. G., female, aged eight years, entered clinic with complaint of hives. Diagnosis was lichen urticatus. During routine examination purpuric lesions about ⅛ to ¼ inch in diameter were noted on the buttocks and lower extremities. A diet diary was kept by the mother. Egg was suspected as the offending food and was removed from the diet. The purpura disappeared and the urticaria also cleared up. Ingestion of egg on several occasions produced purpura but no urticaria. The clotting and bleeding times and also the blood picture revealed no abnormality.

CASE 5.—A. A., female, aged eleven years, was referred from skin clinic on June 8, 1934, because of numerous small pinhead-sized purpuric spots on the legs, thighs, and forearms. There was also an erythematous rash on the chest with a few purpuric lesions. Examination of the child revealed nothing of note otherwise. The mother was advised to return to the clinic four days later with the child, at which time all of the purpuric lesions had disappeared. In giving the history, the mother stated that tomatoes and cucumbers were the only foods recently added to the diet which might have caused the purpura. She was advised to feed the child tomatoes and cucumbers, and within twenty-four hours the purpura reappeared. After removal of these foods from the diet for a couple of weeks they were refed, and the purpuric lesions reappeared. The mother was advised to remove these foods entirely from the diet. Several months later, in the fall of the year, these same foods were given again, but we were unable to reproduce the purpura. This led me to believe that there might have been some other factor involved in the spring of the year which we did not have present during the fall. There possibly may be some relationship between pollens and foods giving a so-called total dose effect during the spring of the year which may possibly account for the high seasonal incidence of purpura in the spring, as noted by Jones and Tocantins.[22]

In the case mentioned above there was no previous allergic history in the child, but the mother had hay fever, and a brother had infantile eczema due to various foods.

CASE 6.—R. Z., aged four years, female, was brought to the clinic for hay fever therapy. She was sensitive to ragweed. (The mother, brother, and sister had hay fever.) Purpura of the lower extremities was noted on numerous occasions. These areas were ecchymotic lesions ¼ to ½ inch in diameter. An elimination diet and diet diary revealed chocolate as the offending food. Lesions were produced on several occasions by feeding chocolate. This child gave a positive reaction to an intracutaneous test for chocolate. No abdominal symptoms accompanied the ingestion of chocolate.

CASE 7.—J. H., male, aged eight years, entered the clinic with a complaint of having had hives for several weeks. Diagnosis was lichen urticatus. Numerous purpuric lesions varying in size from about ⅛ to ½ inch in diameter were noted on both the upper and lower extremities. Bleeding time was 1¾ minute; clotting time, 3 minutes. The platelet count was 360,000. Elimination diet and a diary indicated chocolate as the offending food. Purpura and urticaria disappeared on removal of chocolate from the diet. Purpura was reproduced on several occasions by the ingestion of chocolate.

CASE 8.—A. H., female, aged five years, was brought into office with the complaint of coughing. During routine examination several purpuric lesions were noted on thighs and lower extremities. The mother had noted these frequently but thought at the time that they might have been due to trauma. The mother had hay fever; a sister, aged three years, had eczema. On keeping a diet diary, the mother noted chocolate was the food producing the purpura. Four or five purpuric lesions about ¼ to ½ inch in diameter appeared from twelve to twenty-four hours after the ingestion of chocolate. Lesions have been produced on several occasions by the feeding of chocolate. Mother also volunteered the information that when the child breaks out with purpura, she becomes very irritable and seems to have pain in her muscles like "growing pains." The child has no purpura when she does not eat chocolate. This child demonstrates a sensitivity to chocolate as her only allergic manifestation. The family is definitely allergic.

CASE 9.—W. K., male, aged five years, child was first seen at the age of two months with infantile eczema. The offending food at this time was orange juice,

and the eczema cleared when the orange juice was removed from the diet. When he was three years old, during a routine examination, several purpuric lesions were noted on the face and lower extremities. Because of the previous eczema we approached the case with allergy in mind. The history revealed that the child's face flushed when the mother gave him chocolate milk preparations. Removal of chocolate, however, did not cause purpura to disappear. On keeping a careful diary of foods taken, the mother noted that a combination of foods eaten—rolled oats plus chocolate—produced the purpura on hips and legs. The purpura appeared about twenty-four hours after the ingestion of this combination of foods. Feeding these foods separately did not produce the purpura. We have reproduced the purpura in this child on several occasions with this combination of foods. I have assumed that we are dealing here again with a so-called total dose effect such as occurs in nasal allergy as described by Eyermann.[23, 24]

I have reported only those cases in which I have been able to reproduce the purpura on several occasions after ingestion of specific foods, with at least a week's interval of freedom from lesions during which time the offending food has been removed from the diet.

Diet diary and elimination diets have been the best method of approach in my experience in determining the offending substance if a food is involved in these cases. Observant mothers have been of much more value to me than skin tests in these children.

In working with a group of allergic children and also those that we presume to be nonallergic, it strikes me that I have noted purpura much more frequently in the allergic group. I have, however, no definite figures relative to the incidence of purpura in either group. It may be of some interest to note that in this small series of cases six of the nine patients were females.

SUMMARY

Purpura may be a first manifestation of allergy in children.

It may occur only after feeding a combination of foods or may possibly occur as the result of the ingestion of a specific food plus exposure to an inhalant or pollen.

Purpura due to food ingestion may occur independent of abdominal or joint manifestations.

The extremities, particularly the lower extremities, appear to be the site of predilection for purpuric lesions in most of our cases and in many described in the literature.

Chocolate is a frequent causative food as noted in this group of cases.

Careful history, elimination diet, and diet diary are more important than skin tests in these cases.

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III. TREATMENT OF THE SEVERAL TYPES OF ADIPOSOGENITAL DYSTROPHY IN BOYS

With Particular Reference to the Use of Gonadotropic Hormone From the Urine of Pregnant Women for Their Imperfectly Developed Genitals

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IN 1933, when a sufficient amount of hormone was available for clinical use, I undertook a study of fourteen boys who showed genital underdevelopment either in the form of hypoplasia or cryptorchidism and subsequently demonstrated the effectiveness of injections of the hormone found in the urine of pregnant women (anterior pituitary-like hormone) on the genital systems of these boys. In this series[1, 2] all other therapy was discontinued in order that the effects of this anterior pituitary-like hormone could be evaluated. That this gonadotropic hormone of pregnancy urine produced stimulative effects on the gonads of these male children was evidenced by the fact that the undescended testes increased in size and descended, as a rule, into the scrotum, and in a number of patients testes which could not be felt on examination prior to treatment were subsequently developed by this treatment. It was presumed that this gonadal gland was stimulated by the gonadotropic hormone and then produced sufficient male hormone to cause, in addition, stimulation of the accessory sex organs, of the external genitals, and of some of the secondary sex characteristics. These effects were evidenced by enlargement of the penis, the epididymis, and sometimes the prostate, the development of the scrotal pouch, and the production of pubic hair. While pregnancy urine alone was the only therapy administered in the study of the above mentioned group of patients and while it produced marked results on the genital system, it was not in itself sufficient to produce a "complete" correction of the condition of the patients in my series. This was necessarily so since there were some with other conditions besides underdeveloped genitals: for example, those whose disturbances were due to primary gonadal insufficiency; others whose involvements were due to thyroid underactivity; while still others (though not included in my present study) whose disturbances were thought to be linked up with involve-

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ment of the pituitary gland. It was therefore considered feasible to classify the patients into their respective types before treatment was instituted. The following classification was used and found practical for clinical purposes.*

1. AGD—hypogonad
2. AGD—hypothyroid
3. AGD—hypopituitary, aneoplastic
4. AGD—hypopituitary, neoplastic
5. AGD—hypopituitary, Froehlich

As can be seen from the above, all cases were prefixed with the abreviation "AGD." This was used to designate adiposity and genital hypoplasia and to indicate only adiposogenital dystrophy without reference to its etiology, not as frequently had been done noncritically before to indicate hypopituitarism or Froehlich's syndrome. With this "AGD" as a general term, the classification was then based upon what I believed was the gland which was primarily affected and involved—the thyroid gland, the gonads, or the pituitary.

It is my belief that, while the above classification is not necessarily foolproof, it is, however, an attempt to clarify heretofore vague and indefinite nomenclature.

If the several types in the above classification, and particularly the first two types, are kept in mind, it can be seen readily that, although they all presented adiposity and genital hypoplasia, there were other differences which distinguished one type from another type. These differences were in the nature of skeletal, somatic, and mental disturbances, the differentials of which were given in detail in previous reports.[1-3]

Considering all these factors, I previously treated these two types with different therapy to study their effects. To some, anterior pituitary-like hormone was given and to others, thyroid. By these results reported in previous writings and referred to above, I showed that under anterior pituitary-like hormone treatment, the genitals could be made to respond greatly but that the osseous system which showed retardation could not be made to respond equally. Also, while the patient developed sexually under treatment with anterior pituitary-like hormone, generally neither the feminine body contour nor the obesity could be modified greatly. On the other hand, in the hypothyroid patient treated with desiccated thyroid alone, the obesity and feminine contours usually were corrected, osseous development was advanced and often rapid, and the sexual development progressed; but the sexual progress was in no way comparable to that seen when anterior pituitary-like hormone was given.

*The types listed have been elaborated and slightly modified from the classification given in my previous reports.[1, 2]

The writer, therefore, considered using the present group of nine male children to test the effects of combined therapy, that is, anterior pituitary-like hormone supplemented by dietary restrictions in the AGD—hypogonad patient, or the concomitant use of desiccated thyroid, dietary restrictions, and hypodermic injections of anterior pituitary-like hormone in the AGD—hypothyroid child, in order to see if complete response could be evoked.*

By a complete response I mean the additional responses which otherwise could not be elicited under a single type of therapy, as diet, or desiccated thyroid, or pregnancy urine alone.

The combined therapy attempted was given to achieve as completely as possible the following responses:

1. The enlargement of the testes and their fixation in the scrotum.
2. The development of the penis and scrotum.
3. The production of pubic hair in boys at the age of puberty.
4. The reduction of weight in the obese and the correction of the feminine type of body contour.
5. The correction of somatic deficiencies and mental and osseous retardation present in the AGD patients.

The following cases cited in detail indicate how closely the patients have come to the desired complete response.

CASE 15.—J. L., a white boy, aged eleven years and six months, was referred by his family physician because of genital underdevelopment. His infantile history, as related to teething, walking, and talking, was normal. His work in school was poor; his responses were somewhat sluggish. He was overweight and presented the average normal total height for his age. His lower extremities (lower measurement) were in normal proportion to his trunk (upper measurement).

Both of his testes were the size of small beans. The left testis was in the scrotum; the right testis receded easily into the canal but could be pulled down. The penis was very small and receded into a poorly formed labia-like scrotum.

Roentgenograms of the osseous development were interpreted to show a slight retardation. The sella turcica was normal. The basal metabolic rate was + 3 per cent.

The patient's status was one of adiposogenital dystrophy secondary to a mild thyroid insufficiency (AGD—hypothyroid type).

Hypodermic injections of gonadotropic hormone of pregnancy urine (anterior pituitary-like hormone) were instituted. The patient received 36,000 R. U. in 135 injections supplemented by thyroid therapy and dietary restrictions.

After nine months of treatment the testes had increased in size and remained fixed in the well-formed scrotum; the penis was enlarged and thickened, and some blond hairs appeared on the mons pubis. His body contour and manner became more masculine, and his mentality was improved.

CASE 16.—Me. W., aged nine years and four months, was referred because of general and genital underdevelopment. His infantile history was said to be uneventful. The patient was sluggish and backward in school and was very mischievous. He was overweight, but of normal height and proportions.

*The anterior pituitary-like gonadotropic hormone of pregnancy urine used in this work was supplied through the kindness of Dr. J. J. Durrett and Dr. H. Sidney Newcomer, of E. R. Squibb and Sons.

The two small testes in midscrotal position tended to recede very easily into the canal. His penis measured ¾ inch in length and was buried in a labia-like fold of the scrotum, which was poorly formed.

Roentgenologic studies of his bony framework gave evidence of slightly retarded osseous development. Roentgenograms of the sella turcica and thymic region showed no abnormality. The underdevelopment of the mandible was evidenced by a recession of the chin and the malposition and crowding of the lower teeth. The sixth year molars had not erupted. The basal metabolic rate was – 22 per cent.

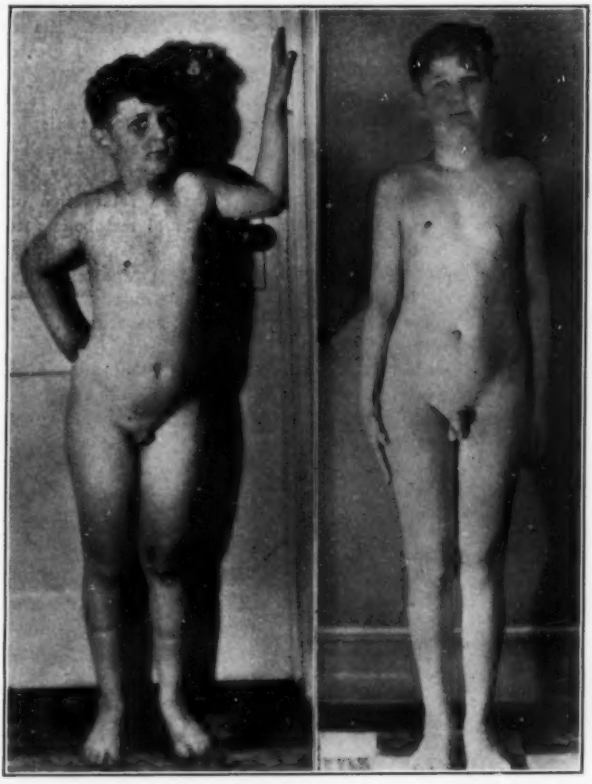

Fig. 1.—Case 16. Me. W., aged nine years and four months (June, 1934) before treatment, and after ten months of desiccated thyroid and seven months of combined therapy (November, 1935). The genital and somatic advance is notable.

A diagnosis of adiposogenital dystrophy, hypothyroid in type (AGD—hypothyroid), was considered, and thyroid therapy was prescribed in addition to hypodermic injections of anterior pituitary-like hormone. Although the injections of anterior pituitary-like hormone were not given, desiccated thyroid was given for ten months. During this period the boy showed general improvement and became more alert although his genital development showed very little apparent change.

After the ten months, when he was ten years old, anterior pituitary-like hormone treatment was begun (in addition to the thyroid therapy). The patient received 22,000 R. U. in sixty-one injections. At the end of seven months of combined treat-

ment, both testes developed to a good size and were fixed in the scrotum. The penis had increased in size, and the scrotum was enlarged and well developed.

His sixth year molars had appeared, and his receding jaw seemed to be correcting itself. He became more alert, showed improvement in his work at school, and was less mischievous. His feminine body contour and his mannerisms became more masculine in type.

CASE 17.—J. G., aged ten years and three months, had a history of a normal infantile development. An examination showed a bright and alert boy. He was overweight; his complexion was pasty; his hair was somewhat dry and coarse; and his skin was slightly dry. There was a complaint of constipation. His total height was the minimum for his age, and the length of his extremities was greater than his trunk.

His right testis was the size of a small shelled almond and was retracted into the inguinal canal but could be pushed down easily; his left testis was smaller and also receded very easily into the inguinal canal. His penis was small and partly buried in the fat of the mons pubis. His scrotum was incompletely formed.

Roentgenograms showed a normal sella turcica and a retardation of one and one-half to two years in osseous development. His basal metabolic rate was – 8 per cent.

A diagnosis of AGD—hypothyroid type was made.

Treatment with anterior pituitary-like hormone was instituted with supplementary thyroid medication and dietary restrictions. The patient received 26,000 R. U. in fifty injections.

At the end of four months of combined treatment there was a marked increase in the size of the testes, which remained fixed in the scrotum. The scrotum developed and increased in size, and the penis, enlarged and thickened, was free from the fat of the mons pubis.

His body contour became more masculine; his pastiness and pallor has diminished; his constipation was corrected; his hair became finer and took on a better luster.

CASE 18.—G. G., aged three years and four months, was referred because of the absence of both testes. The mother stated that her physician felt the right testis high up in the canal at birth. The mother thought that she also felt the right testis during the boy's early infancy. Several careful examinations failed to reveal any definite right testis, but they did seem to reveal an indefinite mushy mass in the region of the internal ring. The left testis was not present in the scrotum or along the inguinal canal, nor was it ever felt. The penis measured ¾ inch in length. There was no semblance of a scrotum.

The child was adipose but not obese. The development of the boy was otherwise normal. His height, 43 inches, was above the normal average for his age (the average normal height at three and one-half years is 37.4 to 40.2 inches). The length of the lower extremities was greater than the normal average for that age, and approximated the length of the trunk. This was a deviation from the normal infantile type and indicated a tendency toward eunuchoid proportions. The osseous development for his age was normal. With the exception of his genital underdevelopment, no other gross physical or mental defects were noted.

A diagnosis of adiposogenital dystrophy with early symptoms of a primary hypogonadism (AGD—hypogonad type) was made.

The treatment and progress of this patient, though still incomplete, is reported at this time. A slightly modified reducing diet was prescribed in addition to the gonadotropic hormone of pregnancy urine.

After 2,500 R. U. of this hormone in a period of six weeks, the right testis could be felt definitely high up at the internal ring, and after 9,500 R. U. in twelve weeks

the left testis could be delineated as a mushy mass at the left internal ring. At the time of this writing, the patient had received 19,275 R. U. divided into sixty-eight injections over a period of five months.

At the end of this period the right testis was felt to be of a good consistency, about the size of a small marble, and could be pushed down to a high scrotal position. With this progress, the right testis was preceded by a fingerlike mass which felt edematous or gelatinous and which also could be pushed down with the testicle *en masse* into the scrotum. The left testis was at this time of fairly good consistency but was situated high up in the canal and occasionally receded into the abdomen. The penis had increased in size, and the scrotal pouch, originally not formed, was developing and becoming globular in shape.

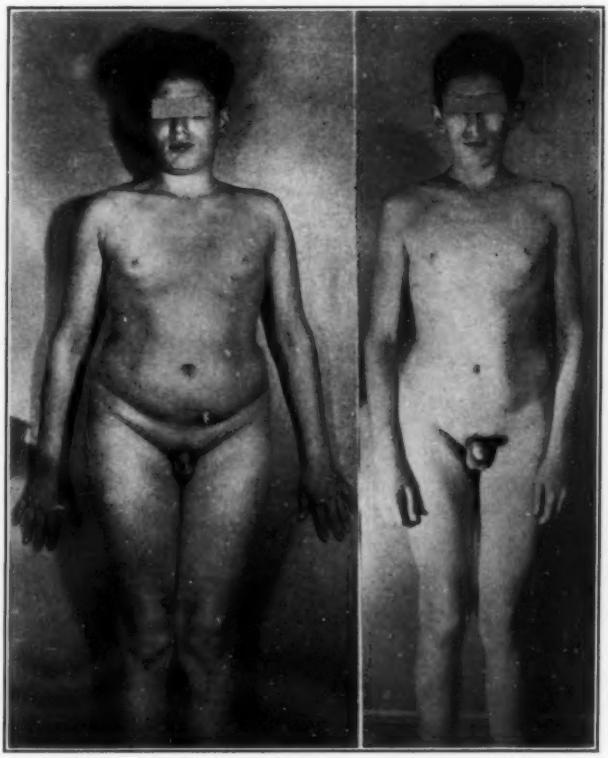

Fig. 2.—Case 19. L. R., aged fourteen years and three months (March, 1935). Note the obesity, feminine body contour and the genital hypoplasia as compared with the more masculine appearance and the genital advance after eight months of combined treatment (November, 1935). The many long black pubic hairs, which appeared after treatment, are indistinct in the photograph.

The boy's height had increased by 1¾ inches in a period of five months of treatment; and although he had gained 6 pounds in weight, this weight was distributed so that he did not present a feminine contour as originally.

CASE 19.—L. R., aged fourteen years and three months, was referred because of suspected undescended testes. The mother stated, however, that when the patient was asleep, she thought she could often see the testes. This boy's infantile developmental history was normal except that he was born prematurely, at seven

months. The patient had always been obese and was able to gain weight very easily. He was childish in his ways, lazy, and mentally slow. Frequently he would go into tantrums when pressed for an answer to a question. On several occasions he was kept back in his grade at school. The teacher told his mother that her boy "seemed to be in a trance all the time." He mixed very little with his companions and preferred to be alone. He had some interests, however. He liked the radio and also showed a preference for drawing.

Physical examination showed a feminine type of body contour. He was 22 pounds above the average maximum weight for his age. His total height was within the average normal for his age, but the relation of lower to upper proportions was eunuchoid.

Fig. 3.—Case 20. L. T., aged eleven years and nine months, before treatment (May, 1935). Note the obesity, the bloated appearance and the external genital hypoplasia as compared with that after five months of treatment with gonadotropic hormone and dietary restriction (October, 1935).

Two testes were in the scrotum, the left the size of a small green olive, and the right slightly larger; both of these were not fixed in the scrotum and receded very easily into the inguinal canal. The scrotum was globular in shape and of fair size. The penis measured 1¼ inches in length and was slightly buried in the mons pubis. Some sparse black hairs were present on the mons pubis.

The basal metabolic rate was – 15 per cent and his pulse rate was 66. The ossification centers and epiphyseal nuclei were well developed for his age; however, the epiphyses of the phalanges and metacarpals were widely open. A roentgenogram of the sella turcica was normal.

A diagnosis of adiposogenital dystrophy, hypothyroid type, was considered.

The patient was treated by dietary restrictions and desiccated thyroid in addition to the gonadotropic hormone of the pregnancy urine. He received 17,000

R. U. of the hormone in seven months. At the end of this period his weight had been reduced by 13 pounds; his height had increased 1¼ inches; his basal metabolic rate had increased to – 1 per cent.

The testes were markedly increased in size and remained fixed in the scrotum. The penis took on a more mature aspect and was 2¾ inches long. The scrotum elongated, and a luxurious crop of black pubic hair was present.

The boy's mentality and reactions were changed. He became more alert, more interested in his surroundings, made much better progress in school; his mother attested that he was a "new boy."

CASE 20.—L. T., aged eleven years and nine months, was referred because of genital underdevelopment. A "thymic status" was suspected by another physician. The patient's infantile developmental history was normal except that at the age of five years the mother stated that the boy received three irradiation treatments for an enlarged thymus. At the age of seven and one-half years the boy was said to have had dropsy and to have passed blood in the urine. He was in bed for sixteen weeks because of this condition.

Upon physical examination, the boy revealed marked obesity and a feminine contour. His weight was 146½ pounds, and his height was 62 inches (the normal

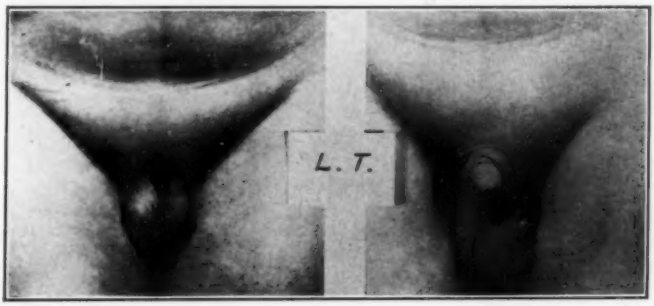

Fig. 4.—Case 20. Close-up of genitals of patient L. T. before and after five months of treatment.

average for his age being from 55.2 inches to 59.0 inches and from 69 to 87 pounds). The relation of the lower extremities to the trunk was eunuchoid in type—the lower measurement being 32½ inches and the upper, 29½ inches. He was bright and alert. X-ray studies showed a normal sella turcica and a normal osseous development. A roentgenogram of his heart showed a slight enlargement in all directions. There was no thymic shadow. The basal metabolic rate was -14.3 per cent. The urinalysis showed specific gravity 1.025; very faint trace of albumin; an occasional hyaline cast, sugar, R.B.C. and pus cells negative.

There were two good sized testes situated in the midscrotal position. The scrotum was well formed. However, the boy's penis was buried completely in the mons pubis and was evident only by a puckering on the surface of the scrotum. When the scrotal tissue was pushed back, the penis measured ½ inch in length. Pubic hair was absent.

A diagnosis of adiposogenital dystrophy of the primary hypogonad type (AGD—hypogonad type) was made.

A strict dietary regime was ordered, and the patient was given an anterior pituitary-like hormone. (This preparation differed from the usual anterior pituitary-like hormone used in this series in that it was extracted from placental substance according to the method of Collip.) Sixty-four cubic centimeters (6,400

biologic day-units) of this substance was given over a period of three and one-half months. During this period of restricted diet and injections of the hormone, the patient's weight was reduced by 31 pounds. His penis increased in size 1¼ inches and emerged from the mons pubis. The lack of the placental extract necessitated a continuation of the treatment with the gonadotropic hormone of pregnancy urine. In the six weeks following, the patient received a total dosage of 3,650 R. U. and showed rapid progress.

The penis developed to a good size, measuring 2½ inches, and presented a more mature appearance. Some sparse long black pubic hairs were present. The testes had not increased in size to any noticeable extent but were fixed in the scrotum.

The patient at this time weighed 113 pounds, having lost a total of 33½ pounds. The feminine body contour was lost. The height had increased 1 inch, and he still showed eunuchoid proportions. He was mentally alert and in good spirits. No thyroid medication had been prescribed for him. Basal metabolic rates taken during the course of treatment were −14.5 per cent after two months, and −28.5 per cent and −19.2 per cent, several days apart, after five months of treatment.

CASE 21.—H. E., ten years and nine months old, was referred because his genitals were not developing properly. His infantile developmental history was normal except that he was obese. The patient was taller than the maximum for his age—height 58¼ inches, weight 106½ pounds; he showed eunuchoid proportions.

The left testis was hypoplastic and was in the scrotum; the right testis was also hypoplastic and was situated high up in the inguinal canal. The latter, however, could be pulled down slightly. The penis was tiny and retracted in a labia-like and imperfectly formed scrotum.

X-ray studies showed normal sella turcica and osseous development. His basal metabolic test was unsatisfactory.

A diagnosis of the AGD—hypogonad type was made.

The patient was placed on injections of anterior pituitary-like gonadotropic hormone. After five months the patient was obliged to discontinue treatment. By this time he had received 8,700 R. U. of the hormone in fifty injections. As a result, the left testis had doubled in size and was fixed in the scrotum; the right testis had descended to a high scrotal position; the scrotum had begun to fill out; and the penis had increased in size.

CASE 22.—S. M., ten years and nine months old, was referred because of an absent testis. The patient's infantile developmental history showed evidence of delayed teething, walking, and talking. He was always constipated. He was somewhat sluggish mentally. Examination revealed a normal sized individual, 19 pounds heavier than the optimum for his age, with a lower measurement slightly greater than the upper. He also showed some localized adiposity, suggestive of the female contour.

His right testis could not be felt along the canal although on external examination of the anterior wall of the right lower quadrant tenderness was elicited on deep pressure, but no trace of a definite mass was noted. The left testis was hypoplastic, the size of a small hazelnut, and was situated in the scrotum, but it receded very easily into the canal. The penis was 1½ inches in length; the scrotum was fairly well formed; and pubic hair was absent.

X-ray studies showed a normal sella turcica; however, his osseous development was two years retarded. His basal metabolic rate was −10 per cent.

A diagnosis of AGD—hypothyroid type was made.

The patient was then given anterior pituitary-like gonadotropic hormone supplemented by desiccated thyroid. He received 17,600 R. U. in sixty-five injections over a period of four months and then discontinued treatment for three months. At the end of three months treatment was resumed, and the patient received an additional 5,000 R. U.

Following this treatment, the left testis doubled in size but was still hypoplastic and in the scrotum; however, neither right inguinal canal nor the scrotum showed any evidence of the presence of the right testis although there were noted some hypertrophied masses of tissue which to touch suggested tissue similar to that of the epididymis. On deep abdominal palpation there was still noted the definite tenderness in the right lower quadrant which was found on the first examination. In addition, superficial palpation revealed a definite globular mass situated in the same quadrant. This mass persisted for several months. It was believed that this mass might be the right testis which was blocked from entering the internal ring. The penis was thickened and had increased in size to 2½ inches.

The patient had grown 2¼ inches in height over a period of nine months; the feminine contour had altered; he was more alert and was doing well in school. (He was placed on the honor roll of his class in the junior high school which he attends.) Hypodermic treatment was temporarily discontinued, but desiccated thyroid was continued. His osseous development at the end of the first six-month period of combined treatment showed advancement.*

CASE 23.—H. U., three years and nine months old, was brought to the office because of frequent acute torsions of an undescended left testis. The boy had a normal infantile developmental history. The patient suffered very frequent acute attacks of torsion with severe pain and on one occasion was rushed to the hospital for operation. This, however, was postponed on account of the cessation of pain and because of the existence of secondary anemia.

At examination he was alert, slightly oversized, but not obese. His lower extremities and trunk were proportioned normally. The sella turcica was normal; the osseous development was somewhat advanced.

His right testis, in the scrotum, was of a fairly good size; his left testis was felt indefinitely midway in the canal, but it could hardly be dislodged from its position. The penis and scrotum were of good size and well formed.

This patient presented no definite or apparent condition of endocrine pathology.

The patient was placed on anterior pituitary-like gonadotropic hormone. He received 6,500 R. U. over a period of two months; then treatment was discontinued for several months. In the interval between cessation of the treatment and its resumption several months later, he suffered the same attacks with the same frequency and severity. Then treatment was resumed for another two months, the boy receiving altogether a total of 14,100 R. U. in forty-seven injections.

The patient's right testis was stimulated but increased in size only slightly. However, the penis was increased in size and showed frequent erections. It was thought that the left testis, then definitely delineated, had descended slightly but still was resident in the canal. The acute attacks of pain which the boy had experienced very frequently prior to treatment had practically ceased during the time of the treatment with the gonadotropic hormone. In spite of this relief from pain during therapy, further treatment temporarily was discontinued because of the temporary priapism that ensued. It is felt, though, that more treatment might be attempted in the very near future.

DISCUSSION AND COMMENT

As a whole, the present group of patients responded well to the therapy in their genital development, mental aspect, and body contour, particularly Cases 15, 17, 19, and 20. The other patients are still re-

*Although the progression of osseous development has not been rechecked yet in some of the hypothyroid patients in this group, I know from my previous experiences and observations of such patients that the addition of desiccated thyroid stimulates the ossification and diminishes the bone lag.[1, 2, 3]

ceiving treatment. Even in Cases 22 and 23, in which the poorest results were achieved, obvious definite gonadotropic hormone stimulation of the genital system was noted.

S. M., aged ten years (Case 22), was of the AGD—hypothyroid type. The excellent response in mental improvement, height, and osseous advance needs no further comment to indicate the benefits of the desiccated thyroid in this case. The anterior pituitary-like treatment in this case caused the left testis to double in size and become fixed in the scrotum and also caused the penis to increase in size. In addition, where originally tenderness was elicited on abdominal palpation in the region of the right lower quadrant, later there could be palpated a definite globular mass which has persisted for several months. This mass is suspected of being the testis, which for some reason has been unable to pass the internal ring. The apparent obstruction at the internal ring, which is concomitant with his glandular dyscrasia, may make operative interference justifiable in the future. Meanwhile, complete glandular therapy is being instituted and will be used until exhausted and found useless.

It is of interest to note in this particular case that upon pushing one's finger up the inguinal canal, even after continued treatment, one felt a rather tight canal and rigid internal ring. In some of the present cases and in others not reported here, in which no testes were felt originally but later developed successfully and descended, treatment with anterior pituitary-like hormone seemed to cause a relaxation of the entire inguinal canal and internal ring so that the examining finger met no resistance and could be inserted with greater ease up the canal and often through the internal ring. There seemed to be maturation, or a sort of "ripening," of the entire inguinal region. In this patient, though, there was no such maturation.

In this connection it is interesting to present the opinion of Spence and Scowen.[4] The latter quote Burrows, who by injection of large doses of estrin into mice produced inguinal hernia and a lax condition of the scrotum and inguinal region similar, I believe, to what might be the condition which I describe as maturation of the inguinal region. In discussing the possible mechanism for the descent of the testes, these authors state that since estrin, as well as testicular hormone, is present in the male, it is conceivable that estrin was produced by stimulating the undescended testicle with gonadotropic hormone and that this estrin played some part in elongating the ligaments and adnexa concerned in the testicle's descent. Whether through the gonadotropic hormonal stimulation of the testes estrin was produced in addition to testicular hormone to cause the maturation referred to in our cases is unknown and problematical. Although to date the evidence so far points only to the fact that the gonadotropic hormone of pregnancy urine stimulates the testes with the probable subsequent

production of testicular hormone to stimulate the sex accessories and secondary sex characteristics, it is very likely that this product of the gonadal stimulation also acts as a stimulus to the entire inguinal region, causing this maturation or "ripening" in preparation for the descent of the testes.

That the mechanism for testicular descent is not primarily mechanical, nor primarily dependent upon the weight of the testes, is suggested by my clinical observations and studies of patients treated with this anterior pituitary-like hormone. I have often noted tiny and hypoplastic testes situated in the scrotum and at other times fairly good sized testes resident in the canal. Whichever may be the case, there is still no evidence to prove what initiates normal spontaneous descent of the testes into the scrotum. Engle[5] suggests that the presence of gonadotropic hormone in the maternal circulating blood during pregnancy probably was involved in the mechanism of descent of the testes in the human. This statement, though interesting, is not as yet final.

In several patients in whom the testes were arrested at the internal ring or were situated intraabdominally (noted in this series in Case 18, which is discussed later), there appeared either before or during the treatment a fingerlike, edematous or gelatinous projection, resembling to touch a hernial sac or hydrocele, which preceded the testes and after sufficient treatment advanced and seemed to plow its way down the inguinal canal. It is known that in the normal mechanism of the descent of the testes in utero there is a bulge of peritoneum, known as processus vaginalis peritonaei, which burrows its way through the anterior abdominal wall and down the inguinal canal into the scrotum. This processus vaginalis acts as a guide in preceding the gonad's transit through the canal into the scrotum. Usually before birth, when gonadal descent is complete, this peritoneal process is in part incorporated into the scrotal lining and in part atrophied. It is plausible to assume, from the observations of my patients, therefore, that this protrusion or edematous mass often referred to as a congenital hernia is simply the processus vaginalis arrested at some point in the descent, so that further progression through the internal ring and into the canal was interrupted, causing the mass to remain at that point. Generally it can be said that this failure to descend probably is due to imperfect and inadequate hormonal stimulus, for, with proper and sufficient hormonal stimulation administered to such patients, the arrested processus vaginalis can again be actuated, and with successful development and complete testicular descent this protrusion or hernial sac disappears.

In Case 18, G. G., intraabdominal testes were suspected. Treatment in this case is still in progress. This case is noteworthy of attention in that this patient with bilateral intraabdominal testes was only three years and four months of age at the beginning of treatment.

Aberle and Jenkins[6] treated two patients of similar age with this anterior pituitary-like hormone, in one of whom there was no response and in the other there was stimulation and descent of the testes into the scrotum.

In Case 18, one testis was definitely absent while the other, though thought to be present on one occasion as an indefinite mass high up at the internal ring, could not be defined after several careful examinations prior to therapy. During treatment with the gonadotropic hormone there soon appeared that gelatinous fingerlike protrusion on the right side, which I have already mentioned. With further treatment this protrusion then descended the canal and preceded, as it did in the other cases, the descent of the testis. This descent occurred in short time and indicated that this right testis was formed and had been ready to enter the internal ring and that it possibly needed only the necessary stimulation offered by the administration of the gonadotropic hormone to cause it to descend. The left testis, too, was felt soon thereafter below the internal ring, but this testis at the time of writing had to be pushed down and held in place before it could be definitely delineated. I believe that this boy will eventually respond completely.

The response of the gonads to treatment in this three and one-third-year-old boy and the response in the case reported by Aberle and Jenkins indicate that the early age of a child is not a contraindication for the institution of this hormone treatment, nor does the early age slow up the response and progress as far as we can judge now. Although one is reluctant to give frequent hypodermic injections to the very young child, nevertheless, if descent or proper development has not taken place within the first two years, hormonal treatment should be considered. The rationale of early hormonal treatment is demonstrated in this young patient. Though he presented no marked gross symptoms of a glandular disturbance (excluding his genital underdevelopment), evidences of a primary hypogonadal nature were already present, such as an accelerated height exceeding the average optimum for his age, an increased lower measurement almost equalling the upper measurement—symptoms usually characteristic of those afflicted with primary gonadal insufficiency—and in addition a feminine body contour and adiposity. Children of this type who have underdeveloped gonads from early infancy and who later, at the time of puberty or thereabouts, show spontaneous descent present, nevertheless, the imprints of this past underdevelopment in the form of somatic and skeletal stigmas. Exactly to what extent such anatomic, skeletal, and often the accompanying psychic stigmas affect the future of these boys is a subject for future study. I feel that those who had received complete treatment in their early childhood probably will not carry these same defects through maturity, for most of these defects can be and

were modified. Whether the beneficial effects evoked from such treatment will be permanent, and whether such conditions as eunuchoid proportions, which remain in spite of treatment, will correct themselves in due time after therapy is only a matter of speculative conjecture.

In addition to somatic and skeletal and often psychic defects resulting from undescended testes, there is too the danger of mechanical complications accompanying cryptorchidism, such as torsion. This is exemplified in Case 23. This patient presented no apparent evidence of endocrine involvement, but he had an undescended testis complicated by a torsion of the spermatic cord. Such complication occasionally accompanies cryptorchidism. It is therefore advisable whenever possible to institute early hormonal treatment in order to avoid this complication. Since in this case the torsion was already present before the institution of treatment, an attempt was made to see what corrections could be made possible with gonadotropic hormone. So far, the torsion has not corrected itself during this treatment, but there has been a marked reduction in the frequency of these acute attacks and in the severity of the accompanying pain. In view of this, more treatment was given to this patient in order to see if lengthening of the cord and descent of the testis could be accomplished. Since, however, there ensued unusual priapism and hypertrophy of the penis, treatment necessarily had to be discontinued temporarily. Further developments in this case will be reported some time in the future.

The effect of treatment with anterior pituitary-like hormone combined with diet, and whenever necessary with desiccated thyroid, was on the whole very successful and produced the benefits without any obvious adverse reactions. The desiccated thyroid was effective to a great extent in the correction of the mental and the general physical make-up of the hypothyroid patients. The anterior pituitary-like hormone was effective in producing gonadal stimulation except in cases in which a mechanical obstruction was present. With treatments kept within physiologic limits, anterior pituitary-like hormone had no deleterious effects on any of the organ systems; it caused no mammary enlargements, no pathologic hirsuties, except in the pubic region if treatment was continued long enough; it caused no undue enlargement of the prostate gland or any urinary disturbances. With continuous large dosage it did, however, cause frequent but temporary priapism. In cases in which osseous retardation was present, it stimulated osseous development to a small extent; this, however, might have been due directly to the genital unfolding that took place. It led to an increase in height greater than the increment expected for a similar period without treatment. Indirectly, anterior pituitary-like hormone caused changes in the psychic make-up of the patients because it caused genital development and made the patients feel normal in this respect and equal to other children.[2]

It has been said that treatment with gonadotropic hormone causes hernias. While it is true that this gelatinous protrusion or sac of which I spoke in Case 18 might be a hernial projection in a strict sense of the word, I feel from my experience in other cases that this protrusion will later disappear after the descent of the testis is complete and that the inguinal canal will tighten up and the internal ring will become rigid. No permanent gross hernias as a result of treatment had been noted in the follow-up of our cases previously reported, nor were they produced as a result of treatment at any time in any of the patients whom we now report.

The dosage, the frequency of injection, and the duration of treatment with the gonadotropic hormone varied as a rule with each individual case and depended upon the desired end-result. The method of procedure was carefully considered in each case. Skill in procedure was acquired only after experience with a sufficient number of such patients.

In a general way my procedure was as follows: 0.5 to 1 c.c. (60 to 125 rat units) of the hormone was given on alternate days for the first few injections. Subsequently the usual dose of 2 c.c. (250 R. U.) was given, as a rule, three times a week. This dose was continued until a congestive skin response was noted in the genital region. When this response was slow in appearing, the dosage often was increased to 500 R. U. and was given three to five times per week. This dose was administered until the desired local skin effect was obtained and then, if found necessary, was reduced to 250 R. U. or less.

The congestive skin reaction of the genitals in my opinion indicated a response of the testes to the stimulation by the gonadotropic hormone. However, a too severe or prolonged congestive reaction of the genital skin and a too rapid hypertrophy of the penis with its accompanying priapism should be avoided before the testes develop properly and become fixed in the scrotum. This can be avoided and the process well controlled, as a rule, by the experienced administrator. The indiscreet use of this hormone, however, may be disappointing. Genital precocity can be produced.

The anterior pituitary-like gonadotropic hormone, however, did not noticeably modify the feminine contour of these boys, nor did it overcome the obesity. These changes had to be made by dietary restrictions and, whenever indicated, by desiccated thyroid. The diet was of a low caloric content (approximately 1,000 calories), high in protein and low in fat and carbohydrate. Supplementary vitamins were provided. The desiccated thyroid used was of a standard brand (Parke, Davis and Company, Armour Laboratories, or autolyzed thyroid of E. R. Squibb and Sons—the latter product not as yet ready for general distribution) and was given in doses varying from ¼ to 1 grain per day.

SUMMARY AND CONCLUSIONS

1. Nine patients ranging in age from three and one-third to fourteen and one-third years with undescended or hypoplastic testes and with hypoplastic external genitals are reported. All of these patients, with the exception of one boy whose glandular status was uncertain, were of the adiposogenital dystrophic type. This term is used here in a general sense to indicate adiposity and genital hypoplasia. The abbreviation AGD was used to designate this adiposogenital dystrophy, and with it was used a further classification to indicate what I believed was the primary offending gland causing the insufficiency, such as the thyroid, the gonads, or the pituitary. The cases in this series here reported were only of the hypothyroid and the hypogonadal types and did not include any of the hypopituitary type. The therapy varied from the gonadotropic hormone of pregnancy urine and dietary restrictions in the AGD—hypogonad patients to the gonadotropic hormone of pregnancy urine, dietary restrictions, and the supplementary use of desiccated thyroid in the AGD—hypothyroid patients.

2. Criteria are presented for what I believe comprised a complete response in both of the above mentioned types.

3. Unusual complications, such as torsion, mechanical obstructions, and hernia, are briefly discussed. It was apparent that the congenital hernia—whether potential or markedly noticeable—which accompanied an undescended testis situated intraabdominally or at the internal ring, could be corrected when the testis becomes resident in the scrotum through the stimulation of the gonadotropic hormone.

4. The question of whether the descent of the testis was mechanical or hormonal is considered. I believe that while we were not aware as to what initiated the normal descent of the testis, in cases in which there had been delay in descent, this descent could be initiated by hormonal stimulation and the whole inguinal region could usually be brought to maturation in the male child through the influence of this same hormonal stimulus.

5. The value of combined therapy was shown in that not only was genital development stimulated and genital maldevelopment corrected, but also many of the other accompanying deficiencies of a skeletal, somatic, or mental nature usually could be improved and redirected toward the normal.

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1176 EASTERN PARKWAY

MESENTERIC THROMBOSIS

In a Case of Chronic Mastoid Suppuration

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THE following case of thrombosis of three branches of the superior mesenteric artery and corresponding veins is of interest in that the condition apparently developed as a result of sepsis in a case of prolonged mastoid suppuration. While mesenteric thrombosis may occur during the course of infectious diseases, such as typhoid fever, scarlet fever, and diphtheria, it most commonly develops in cases of ulcerative endocarditis. In the present case, however, clinical as well as postmortem findings excluded endocarditis as a factor.

As Ross[1] states, the superior mesenteric artery, which is said to be an end artery, alone supplies the small intestine and practically all of the large bowel with the exception of the descending colon, sigmoid, and rectum. The duodenum has a double blood supply. The superior mesenteric artery is much more frequently the seat of thrombosis and the condition should be much more serious than if the inferior mesenteric, which is not an end artery, is involved. But in either location the condition is of such gravity that recovery is extremely rare. Arterial block in the mesentery is far more common than obstruction of the venous circulation. The symptomatology is the same whether thrombosis or embolism is present and even at operation or autopsy it is difficult to determine in a given case with which we are dealing. Venous conditions are thrombotic, while arterial obstruction may be due to embolism or thrombosis; or thrombosis may develop at the site of lodging of an embolus. Thrombosis results in a stoppage or diminution of the blood supply of a certain segment of the bowel. Peristalsis is slowed and later stopped, resulting in a local paralytic ileus with signs of intestinal obstruction. Later gangrene or perforation may supervene. Pain and constipation are prominent symptoms; the melena, occasional hematemesis, and the presence of a primary disease in the heart or abdomen differentiate these conditions from acute intestinal obstruction. Acute meteorism results with all the signs of intestinal obstruction. Gangrene or even perforation develops in the segment affected, rapidly followed by peritonitis.

E. G., aged six years, white, female, was admitted to the Montefiore Hospital, July 30, 1934, with a complaint of gastric pain and hemorrhage. The past history was of interest only in the fact that at the age of one and one-half years, she developed a bilateral suppurative otitis media; bilateral mastoiditis developed; and operation was performed on both mastoids. The mastoid wounds never healed and continued draining. For two months prior to admission to the hospital she had complained of abdominal pain, later related to taking food. Three days before admission she had vomited and had become very pale. On July 29, 1934, she vomited blood

clots and had a dark, tarry stool. Examination on admission showed a poorly nourished girl with pale yellowish skin. The pupils were slightly dilated; the conjunctivae showed no jaundice. There was no hemorrhage from the nose, mouth, or throat. Thyroid not enlarged. Both mastoids were draining purulent material from large sinuses extending into the antrums. The heart was slightly enlarged; a systolic murmur was heard at the pulmonary area. The abdomen was retracted, showed no visible peristalsis, or no localized tenderness or rigidity; peristalsis was heard well over whole abdomen. The spleen and liver were not enlarged. There was no purpura or other skin eruptions; the joints were normal. The impression was that there had been a hemorrhage from a gastric or duodenal ulcer or possibly from esophageal varicosities.

Laboratory Data.—The urine showed a faint trace of albumin and a trace of sugar; microscopic examination was negative. On admission the blood showed hemoglobin, 27 per cent; red blood cells, 1,610,000; white blood cells, 19,000; polymorphonuclears, 84 per cent; lymphocytes, 16 per cent; platelets, 350,000; no immature red blood cells, poikilocytosis, or anisocytosis. Coagulation time was two and one-half minutes; bleeding time, ten minutes; retraction of clot, thirty minutes. Fragility test showed that hemolysis began at 0.38 per cent and ended at 0.30 per cent. Blood calcium concentration was 11.2 mg.; blood culture was sterile. Smears from the ears showed pneumococci, staphylococci, and streptococci. The highest temperature was 102° F.; pulse, 90 to 150; respirations, normal. Following treatment, the hemoglobin content increased to 48 per cent and the red blood count to 4,440,000. Intravenous transfusion of 300 c.c. of citrated blood and later coagulen were given. Continuous venoclysis of 5 per cent glucose was instituted. Hypodermics of morphine and atropine were given as indicated. In surgical consultation (Dr. T. A. Schubb) operation was not advised. Following the transfusion the patient improved; there was no further vomiting, hemorrhage or abdominal pain. Bismuth subcarbonate was given by mouth; iron and arsenic hypodermically. X-ray examination of the gastrointestinal tract showed no pathology. The patient was discharged from the hospital Aug. 9, 1934.

She was readmitted Sept. 6, 1934; on the previous day she had developed high fever and chills with diarrhea, vomiting, and abdominal pain, which gradually increased in severity. Marked pallor, weakness, dehydration, and loss of weight were noted. The heart was rapid, slightly enlarged, with no murmurs. Fine moist râles were found at both bases of the lungs but no dullness. Abdomen was retracted; there was no visible peristalsis, no localized rigidity or masses; good peristalsis was heard over whole abdomen. No purpura or joint involvement was noted. The temperature ranged from 100 to 106° F. the first day in the hospital; then it was of a septic type, as high as 102.6° F. A diagnosis of acute septic infection from old mastoid suppuration was made. Sept. 10, 1934, after a period of slight improvement, a profuse intestinal hemorrhage occurred following a marked ballooning of the colon with visible peristalsis; the abdomen then became soft; the bowel was no longer visible. Her general condition became poor; the pulse became very rapid. It was believed that the patient had had a hemorrhage from an ulcerative process in the colon. In surgical consultation with Dr. T. A. Schubb, operation not advised since the process in the bowel appeared to be too widely generalized to be helped by any surgical procedure and, especially, in view of the fact that the patient was septic and had signs of pulmonary involvement. Proctoscopic examination was negative except for the presence of blood in the bowel. On Sept. 11, 1934, she had another bloody stool following a recurrence of the marked ballooning of the bowel. The lungs showed hypostatic congestion at both bases, and the heart became much enlarged. On Sept. 12, 1934, there were several attacks of hematemesis and intestinal hemorrhage with edema of the lungs. Death occurred at 9:44 P.M.

Laboratory Data.—The urine showed traces of albumin, no sugar, 8 to 12 white blood cells per high powered field, heavy and fine, coarse and hyaline casts. On ad-

mission, the blood findings were hemoglobin, 80 per cent; R.B.C., 4,400,000; W.B.C., 7,700; polymorphonuclears, 67 per cent; lymphocytes, 33 per cent. On the day of death hemoglobin 59 per cent, R.B.C., 2,690,000; W.B.C., 6,000. Blood culture was sterile; culture from mastoids showed staphylococci, and streptococci; stools, blood positive; negative for tubercle bacilli.

Autopsy Report.—Both mastoid regions showed deep fistulous tracts draining the sinuses, the openings of which were surrounded by unhealthy granulation tissue. *Esophagus* showed no ulcerations, varicosities, or bleeding. *Chest* showed hypostatic congestion at both bases but no gross evidence of pneumonic changes or tuberculosis; no pleural effusion. Peribronchial lymph glands were normal. *Heart* was normal in size and shape. Pericardium was smooth and shiny, containing only the normal amount of serous fluid. The endocardium and valves were intact; no anomalies were noted. *Abdomen* showed moderate increase of serous fluid, no evidence of peritonitis; marked bluish discoloration more intense in the jejunum than in the ileum or colon; there was marked distention of these parts (Fig. 1). The stomach wall

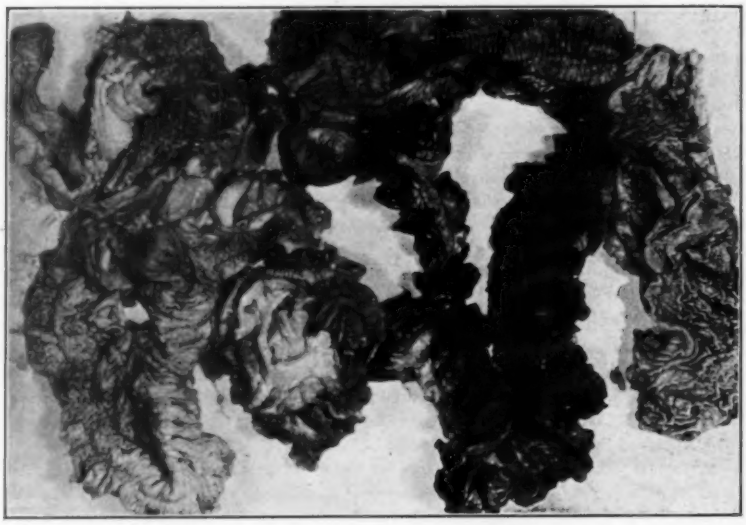

Fig. 1.—Gross appearance of the intestines; gangrenous involvement of the jejunum.

was greatly distended and thinned out, containing about 500 c.c. of dirty coffee ground fluid. The mucosa was smooth and glistening, with no evidence of ulceration. The pyloric sphincter was freely open. The mucosa of several loops of the jejunum showed marked congestion and purplish discoloration; the wall was edematous and gangrenous, but no gross ulcers were found anywhere in the bowel. The mesenteric lymph nodes were unusually large, soft, and pinkish. Three minor branches of the superior mesenteric artery were found to be thrombosed by firm clots; the corresponding mesenteric veins were also thrombosed. The spleen was slightly enlarged.

Anatomical Diagnosis.—Massive intestinal hemorrhage (gangrene of the jejunum); superior mesenteric thrombosis. Bilateral chronic mastoiditis with fistula. Congestive terminal pneumonitis. Profound anemia. Septic splenitis. Mesenteric lymphadenitis.

Sections from the intestines showed gangrene of the loop of jejunum (Fig. 2) due to thrombosis of the mesenteric vessels (Fig. 3). Sections of the lymph nodes re-

vealed no evidence of tuberculosis or malignancy. Sections of the lungs showed complete obliteration of the alveoli with hemorrhage. The bronchioles were filled with exudate. Sections of the heart, pancreas, and liver showed no pathologic changes. The spleen showed hyperplasia of the malpighian corpuscles. In the kidneys there was obliteration of the space in Bowman's capsules and diminished vascularity of the glomeruli. The tubules showed cloudy swelling.

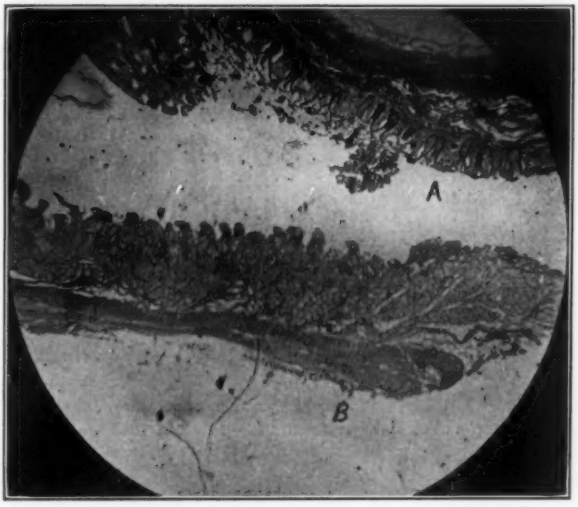

Fig. 2.—*A*, practically normal mucosa and, *B*, section from gangrenous area of jejunum showing complete necrosis of the mucosa and, to a certain extent, of the muscular layers (magnification ×50).

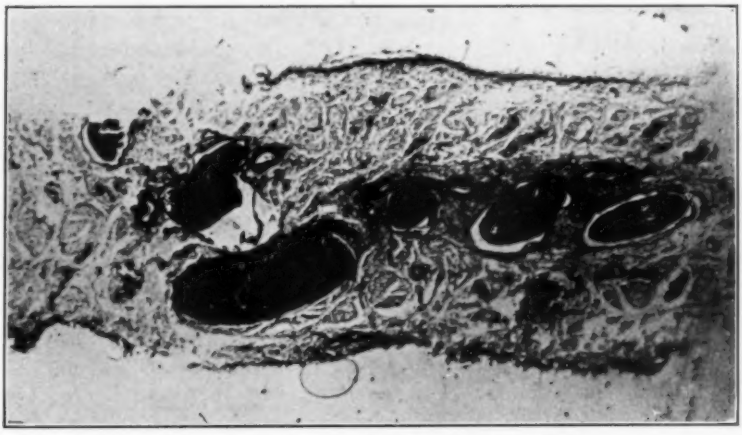

Fig. 3.—Low-power magnification of thrombosed mesenteric vessels.

TREATMENT

Daily blood transfusions and continuous venoclysis of 5 per cent glucose solution were given. Coagulen was given intravenously and

calcium gluconate, intramuscularly. Hypodermics of morphine sulphate and atropine were given to quiet peristalsis; coramine and caffeine sodium benzoate, for stimulation as indicated. Bismuth subcarbonate was given by mouth in teaspoonful doses every three hours. The foot of the bed was elevated. Surgical treatment was deemed inadvisable; and had an operation been performed, it would have been impossible to have localized the diseased part of the bowel, as it was only after repeated washing of the bowel postmortem that the affected part was determined. The entire small and large bowels appeared dark blue due to blood staining of the intestinal wall. Ordinarily, early operation and resection are indicated if the vitality of the intestine has not been gravely affected. If the patient's condition contraindicates resection, the gut should be drawn out of the abdomen, fastened to the edge of the wound and Paul's tube introduced, resection to be performed later. Spinal anesthesia is preferable. Unless operative resection is possible, death is inevitable, and in septic cases such as the one here reported, it is practically inevitable in any event.

COMMENT

How great a part the chronic mastoid suppuration played in producing the mesenteric thrombosis is difficult to estimate. There was no clinical evidence of sinus thrombosis; emboli from this source could not reach the mesenteric arteries unless there was a patent foramen ovale. Unfortunately, permission to examine the head and neck more fully at postmortem examination was refused. It is probable that the thrombosis developed during the last few days of life; the postmortem examination showed that the gangrenous condition was not very far advanced. Slowing of the circulation in the arterial branches and injury to the intima from sepsis or anemia are possible factors. The negative blood culture would not necessarily rule out a blood stream infection resulting from the mastoid suppuration. The blood examinations would definitely exclude any form of blood dyscrasia. The postmortem examination did not reveal any condition in the stomach that might have been responsible for the gastric hemorrhage on the first admission to the hospital.

SUMMARY

A case of thrombosis of three branches of the superior mesenteric artery, complicating an acute septic process in a patient with prolonged chronic mastoid suppuration is reported, with a discussion of the pathology, symptomatology, treatment and postmortem findings.

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THE PROBLEM OF DENTAL CARIES WITH RELATION TO BACTERIA AND DIET

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DURING the last few years we have seen a wholesale attack upon the problem of dental caries by a host of workers, the fruits of whose labors seem to be characterized chiefly by a mass of conflicting data. This observation is not made in a spirit of pessimism since it is very possible that this material, as chaotic as it now seems, may provide a valuable clue toward the ultimate solution of this perplexing problem. Some of the present confusion in dental caries research is in a measure due to the rapid advances made in the field of nutrition. Some of these contributions were invaluable to medicine but not so applicable to the caries problem.

The trend in dental caries research has naturally been influenced by developments in allied fields. Miller[1] made his important observations soon after Pasteur and Koch's bacteriologic investigations had opened vast fields in medical research. In the years that followed, autstanding contributions were made in caries research by Goadby,[2] Kligler,[3] Howe,[4] Baumgartner,[5] McIntosh,[6] Rodriguez,[7] Clarke,[8] Maclean,[9] and others, leading to a generally accepted concept of the etiology of dental caries. It was agreed that the carious lesion resulted from the action of acid upon the tooth. The acids in turn were produced by acidogenic bacteria which became localized on protected tooth surfaces.

Then the world became vitamin conscious. It soon was common lay knowledge that scurvy is caused by a diet deficient in vitamin C, that rickets may be cured by feeding vitamin D, and that pellagra and even some anemias result from deficient diets. The question then arose: Why not dental caries? Consequently, May Mellanby[10] explored the vitamin A and D deficiency possibilities and became convinced that both substances were requisite for caries prevention. Howe[11] was equally certain that vitamin C was the anticaries factor, while others hoped to control the disease with diets well fortified with vitamin D.

A diet including the corrective factors considered essential by Mellanby,[10] Howe,[11] Hanke,[12] Agnew,[13] Hawkins,[28] Boyd and Drain,[14] Klein and McCollum[15] would contain optimum amounts of calcium and phosphorus, the least possible amount of cereal, a liberal supply of butter and eggs, cod liver oil, and at least a pint of orange juice a day. Still, the smallest amount of caries among children reported in recent years occurred in an institution where the diet was quite markedly deficient

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in all these respects and unusually high in cereal.[16] The conclusions made by the above named investigators were founded on careful, laborious observations. There can be no doubt that each group actually observed a decreased amount of caries on the dietary programs suggested by them. But, still, their conclusions are inharmonious. It would seem either that dental caries is due to a number of things or that one factor was common to all of these procedures if caries was actually prevented in each instance. The latter possibility may be found in the sugar content of the various protective diets. It is true that sugar has not been emphasized by the above listed authors, but all of them advise against excessive amounts of candy, and Boyd and Drain first observed arrested caries in children on diabetic diets. In the institution cited by us in which caries was so rare, candy was completely forbidden, and artificially sweetened foods were seldom used.

We have very little evidence that caries of the teeth is a mineral deficiency disease. Analyses for calcium and phosphorus by Hanke,[12] Karshan,[17] and Hubbell and Bunting[18] in our own laboratories revealed no significant differences in the blood and saliva of caries-susceptible and caries-free individuals. Boyd and his associates[19] noted an increased mineral retention in the caries-free group, but this was not confirmed in our own laboratories.[20] Furthermore, no mechanism has been satisfactorily demonstrated by which calcium or phosphorus may be incorporated in or withdrawn from the enamel of fully erupted teeth by way of the blood stream. It is such inconsistencies as these that lead us to search for some extrinsic etiologic factor in dental caries which might be influenced by diet or by some constitutional state.

It is well known that enamel is readily soluble in acid and that the aciduric bacteria in the mouth are a potent source of acid production. These organisms, furthermore, have the faculty of becoming attached to the enamel in out-of-the-way places, inaccessible to the toothbrush or the excursions of food during mastication. In fact, they become localized in just the areas where carious lesions are likely to occur. Work of Goadby, Howe, Kligler, Rodriguez, McIntosh and our own work point very strongly to the lactobacilli in the mouth as the most apparent causative factor. These organisms not only produce an abundance of acid but also continue to thrive in the acid environment which they create. In this respect they are more suitable for caries production than the mouth streptococci, which do not survive in so acid a medium. The streptococci can be recovered with ease from the saliva regardless of the state of caries activity, whereas the lactobacilli although plentiful in susceptible patients are only occasionally isolated from caries-free individuals. We have also found that the lactobacilli are not secondary invaders since they may be demonstrated in the mouth from six months to a year before there is any clinical sign of caries. This observation has been confirmed by Enright and Friesell.[21]

Boyd and Drain relegate bacteria to a position of minor importance in dental caries, and they consider the lactobacilli no more significant than the other acid-producing bacteria in the mouth. This is in agreement with Hanke and Tucker.[22] On the other hand, Clarke and Maclean attribute great etiologic significance to bacteria but are of the opinion that American observers have overemphasized the importance of the lactobacilli. These British investigators are convinced that *Streptococcus mutans* is the organism most responsible for the destruction of the tooth. They have been able to recover these organisms consistently from the deeper layers of dentin in carious teeth. Their observations are based on cultures taken from extracted teeth. The tooth is first split and the lesion approached from the pulpal aspect, and in this way they found that the streptococci had penetrated farther toward the pulp than the other organisms usually found in carious dentin. This may or may not be significant since the bacteria are drawn into the dentin in the wake of the decalcifying acids, and we know that the dentin may be generally carious before the enamel, which is greatly altered, has become disintegrated. If this is true, the acids which first attack the dentin are produced by the bacteria on the enamel. As the destruction of the enamel becomes more complete, a variety of mouth bacteria are drawn into the dentinal tubuli toward the pulp. It would seem that bacteria associated with the initial stages of caries are of greatest importance. Most investigators, including Clarke and Maclean, are in agreement that the lactobacilli predominate in early enamel caries. That being the case it seems advisable to concentrate on these organisms, for surely the best way to prevent caries is to prevent the initial attack.

In our earlier bacteriologic investigations cultures were taken from tooth scrapings and grown in glucose acid broth. The acidity of the medium eliminated the contaminating factors and the sought-for organisms were often procured in pure cultures. Very frequently, however, acid-producing streptococci and yeast grew in this medium as well. The most striking feature of our findings was the decisive difference between cultures taken from susceptible and caries-free individuals. Although, as stated above, caries cultures were often complicated by the presence of yeasts and streptococci, the cultures from caries-free mouths contained streptococci and sometimes yeasts but no lactobacilli. It has long been believed that these organisms normally inhabit all mouths. It is true that they may be found in most mouths just as caries is found in most mouths, but there are a favored few who escape this troublesome affliction and to them the lactobacillus is foreign. It is remarkable that these individuals may mingle freely with their fellow men, most of whom are heavily infected, yet they themselves resist infection. This is particularly extraordinary in children who are so continuously exposed to salivary contamination during their normal contacts with other children. It is erroneous to assume that the lactobacilli are normal to all mouths.

We have also found that they are not uniformly distributed on all the teeth in the mouth. Out of a series of scrapings taken from the various exposed surfaces of all the teeth in a single mouth, it is not uncommon to obtain but one or two positive cultures, despite the fact that the saliva may be heavily infected at the time. It is because of this condition that we and others occasionally failed to demonstrate the presence of this organism in months that subsequently developed caries. For this reason the practice of culturing tooth scrapings was replaced by an examination of the saliva activated by the chewing of paraffin. By this method[23] we are able to estimate the relative number of organisms per sample of saliva. In the majority of cases we have found a relationship not only between the occurrence of the lactobacilli and the incidence of caries but also between the degree of caries activity and the abundance of the organisms as well. Since the saliva becomes infected with the lactobacilli several months before the appearance of the carious lesion, it is possible by bacteriologic methods to predict caries activity with surprising accuracy.

Many do not agree that a causal relationship exists between the lactobacilli and dental caries. Regardless of this controversial point, these organisms afford an excellent means of estimating caries activity and in this capacity are invaluable aids in research. If an arrest of caries is imminent, the acidophilus count is bound to drop off far in advance of any clinical sign. The reverse is also true, for in increased caries activity the bacteriologic count mounts at least four months before new lesions can be demonstrated. Long, costly caries prevention experiments can be cut short or modified if the desired bacteriologic change does not take place within a reasonable time. The total disregard of bacteriology in caries experimentation is sheer extravagance. This does not mean that the efficacy of a caries prevention program is to be judged solely by the change in the oral flora. Bacteriologic examinations are highly informative, but the final test must, of course, be clinical. Caries cannot be said to have been successfully checked unless no new lesions appear and existing cavities grow no larger for at least a year. This necessitates a most rigid comparative clinical check-up at the beginning and end of the period of observation.

Those who have discounted the importance of the lactobacilli in caries have done so for one or more of three reasons. First, some have been led to ignore these organisms because they may be demonstrated in mouths of individuals who are free from caries. In each instance this conclusion has been drawn from one or two bacteriologic examinations made over a period of from five months to a year, and failure to take into consideration the fact that *L. acidophilus* as well as any other organism may appear in any mouth sporadically regardless of its clinical state. Only rarely has this organism been demonstrated to be consistently present in a mouth in large numbers for over six months without the ultimate ap-

pearance of a carious lesion. In order to demonstrate the relationship of these organisms to dental caries, bacteriologic examinations must be made at weekly or monthly intervals over an extensive period of time.

Second, some investigators claim that they have been unable to isolate *L. acidophilus* from carious lesions. This can be explained on the ground that not all open carious lesions are active. A lesion can be considered active only if it has progressed after a lapse of several months. In only one case out of the thousands we have observed, have we failed to isolate the lactobacillus from a lesion which had progressed in size subsequent to our examination. The presence or absence of these organisms in a cavity is an excellent aid in determining whether or not the process is active. The most recent publication in this country in which the author states that he failed to recover acidophilus from caries was based on but a single bacteriologic and clinical examination. It is impossible either by bacteriologic or clinical methods to diagnose caries activity by one examination.

The third argument advanced against the acidophilus thesis is an outgrowth of some of our own publications, the significance of which seems to have completely escaped some observers. This has been most recently exemplified by a statement in a recent monograph by Gay and his associates[24] in which it is stated that we have failed to produce caries by feeding acidophilus culture; therefore, these organisms are not the cause of caries. As a matter of fact, our acidophilus feeding experiments have taught us that any immunity which one may possess against caries seems to be directed against the lactobacilli. These organisms are apparently unable to grow in the saliva or to gain a foothold on the tooth during caries immunity. We have consistently failed in the attempt to implant the lactobacilli in the mouth of any individual who had never had caries.[25] Since some human beings reach maturity without ever having had caries, it is not unthinkable to suppose that they may possess a type of immunity to this disease. It was in this type of individual that we failed to produce caries by feeding acidophilus cultures. Repeated saliva as well as stool cultures of these individuals failed to reveal these organisms, even after intensive dosage of *L. acidophilus* cultures. This suggests the possibility that immunity to dental caries may be a systemic quality, as a result of which the *L. acidophilus* is prevented from growing in the mouth and intestinal tract. Our study of this condition led to some very interesting observations. It was found that many of these individuals, 70 per cent to be exact, reacted positively to intradermal inoculations of *L. acidophilus* filtrate[26] as evidenced by a local erythema within eight hours at the point of injection. It was subsequently observed that the same proportion of caries-free individuals contained *L. acidophilus* agglutinins in the blood serum, whereas 70 per cent of the susceptible persons tested yielded negative skin tests and contained either no demonstrable acidophilus agglutinins or else agglutinin titers

which were much lower than was ordinarily found in the caries-free. We have also reported that the agglutinin titer of a susceptible person may be temporarily raised to the level of a caries-free titer by administration of a polyvalent acidophilus vaccine. This change was also accompanied by a change in skin reactivity from positive to negative. We were seriously handicapped in this work by the formation of rather severe abscesses at the point of introduction of the vaccine. This was later found to be due to the presence of R forms of acidophilus in the vaccines. On the other hand, vaccines made up entirely of smooth cultures failed to induce agglutinin formation in the humans under our observation. It is well known that both the R and S suspensions of acidophilus are antigenic in the rabbit, but it is possible that this is not so in the human. This statement is not meant to carry a note of finality since conclusive evidence would require a much more impressive number of attempts than we have made up to this time. We will, of course, refrain from subjecting humans to our vaccine until some means is found of removing its irritating qualities.

This may be a good juncture at which to introduce another matter which has proved exceedingly irritating in some quarters, and that is the terminology of the particular lactobacillus upon which our attention has been centered for these past ten years or so. It has been said that *Lactobacillus acidophilus* is native to the intestinal tract and that the organism which we associate with dental caries is not *Lactobacillus acidophilus* at all but rather a lactobacillus which is peculiar to the mouth. It was therefore suggested that we speak of the caries organism as *Lactobacillus bucallis* or some name that would distinguish it from the forms found in the intestinal tract. We have found it difficult to concur with this view since we have found that both the oral and intestinal strains possess the same general characteristics, and we have isolated the so-called intestinal types from the mouth and the caries type from the intestinal tract. Culturally, we find no greater differences between the mouth and intestinal types than exist between the various types of the supposedly intestinal forms. Most workers in the field agree that there is not sufficient variation between the lactobacilli of the mouth and intestine to warrant separate classifications. This is of more than academic interest since those interested in the acidophilus milk industry are naturally anxious not to have the name of their product associated with the possible cause of dental caries. It is unfortunate that this situation should exist since we have no reason to believe, nor have we ever stated, that the taking of therapeutic doses of acidophilus milk will in any way influence the course of caries in a susceptible individual, and we also have reason to suspect that the milk may be safely taken in exaggerated quantities during immunity without any likelihood of the organisms becoming implanted in the mouth or, indeed, in the intestinal tract.

Although we have failed to implant the lactobacilli in individuals who had never had caries, we found that we could increase their numbers in susceptible subjects by increasing the carbohydrate in the diet. We have reported from time to time our belief that sugar was perhaps instrumental in stimulating caries activity. During the last few years we have found experimental evidence which strengthens this view. Koehne[27] reported a striking correlation between the carbohydrate content of the diet, the occurrence of *L. acidophilus,* and the incidence of dental caries.

This study was conducted in the University of Michigan Hospital on twenty-three girls ranging from six to thirteen years of age. These girls were metabolically normal and were under complete dietary supervision, their daily food intake being weighed at each meal. Nine out of fourteen of the girls, when placed on a basal diet which was nutritionally adequate but in which the amounts of sugar and sweets were minimal or allowed only in moderate amounts, showed an apparent arrest of dental caries after several months. The other five showed but slight caries activity. In these five the acidophilus counts remained high, whereas in the nine in whom caries was arrested the organisms either disappeared entirely or were present in small numbers.

Thirteen children were put on the same basal diet plus 100 grams or more of candy per day. Within a few months nine of them showed increased caries activity and high salivary acidophilus counts. In the remaining four, caries was inactive, and the acidophilus count was low or negative. In none of these cases was the candy consumed at the expense of the more essential constituents of the diet since all of the children ate their full, regular ration in addition to the excess carbohydrate. Three of the four girls in whom caries activity remained unstimulated were distinct caries immune. They were of the type spoken of above in whom we had never been able to implant *L. acidophilus.*

The growth of acidophilus in the mouth is extremely sensitive to the manipulation of carbohydrate in the diet, as evidenced by the rapidity with which the change in the flora of the saliva takes place. In many individuals the reduction in the acidophilus count takes place within forty-eight hours after the adoption of a sugar-free diet. In some instances it is necessary to withhold starch as well as sugar in order to bring about the desired change. Conversely, the counts are increased tremendously in many cases within twenty-four hours after the change from a moderate to a high sugar diet.

Recently we were able to feed large amounts of candy to children in the institution mentioned above, in whom dental caries is remarkably low and who received artificially sweetened delicacies rarely. This study was conducted on fifty-one children who had been under our observation for a number of years during which time complete records were kept of the incidence of dental caries and the behavior of *L. acidophilus.* As stated previously, our periodic clinical examinations revealed very little

active dental caries in this institution despite the fact that the diet employed was nutritionally inadequate. For five months fifty-one of these children were provided with all the chocolate creams, fudge, and hard candies they could eat. Most of them enjoyed participation in the experiment and consumed enormous quantities of candy ranging from three to four pounds a week. After the first month, the candy intake dropped off considerably, but most of the children managed to eat some candy daily.

During the twelve-month period previous to the onset of the candy-feeding experiment, seven of the fifty-one children showed some signs of caries activity. At the end of the five-month experimental period during which the candy was eaten, a clinical examination revealed new caries in twenty-three, which finding showed an increase of 31 per cent in the comparatively short period of five months. Of greater significance than the clinical findings was the change in the salivary flora produced by the candy consumption. Within twenty-four hours after the first allocation of candy, it was evident that a marked change was taking place. Many of the cases which had been consistently negative became positive for *L. acidophilus* for the first time. Before the end of the experimental period there was an increase in the acidophilus content of the saliva of 80 per cent of the children. Within three weeks after the candy was discontinued, most of the counts returned to approximately the pre-candy level, and a clinical examination five months later revealed no new additional caries.

We have produced considerable evidence in the past to show that an increase in the acidophilus content of the saliva was followed by active dental caries within a year in 85 to 90 per cent of cases. Within the short period of this experiment an increase in acidophilus was followed by active caries in 44 per cent of the children. It is reasonable to assume, therefore, that if the experimental period had been prolonged to a year, a greater increase in caries activity would have resulted. This does not prove conclusively that the consumption of large amounts of candy will increase dental caries activity, but it does point rather convincingly in that direction.

It is true that the problem of dental caries is still unsolved, but certainly the atmosphere is becoming somewhat clarified. We have been a bit sidetracked by the revolutionary contributions made by nutrition. It is becoming quite evident that caries susceptibility is not determined by the structure of the tooth. The rate of caries destruction is very likely influenced by the nature of the tooth, but we have ample evidence to state definitely that under favorable environmental conditions, the most resistant teeth become carious and poorly calcified, hypoplastic teeth remain caries-free if conditions favorable to caries production are not present.

There is considerable evidence that these conditions are reflected in the character of the oral flora. If the lactobacilli are numerous, caries will invariably follow, regardless of the quality of the tooth structure. The conditions which determine the ability of these organisms to survive are evidently not influenced by the calcium, phosphorus, or vitamin content of the diet. A protective diet, so far as caries is concerned, need not even be nutritionally adequate if it is low in sugar. In most cases the oral lactobacilli may be inhibited by the depression of the carbohydrate in the diet, although some individuals who seem to be hereditarily immune to caries remain free of these organisms regardless of the amount of sugar consumed. We are as yet unaware of the nature of this protective factor except that it has a very striking effect upon the welfare of the lactobacilli.

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THE DETERIORATION OF COD LIVER OIL AND THE DIET OF CHILDREN

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MOST pediatrists at the present time believe that the diet of children, and especially that of infants, should be fortified with some source of vitamins A and D. Cod liver oil has for many years been the vehicle of choice for administering additional fat-soluble vitamins. Within recent years, however, many other substances, including other potent fish liver oils, have been added to the list of readily available concentrated sources of the vitamins. Since vitamins A and D are soluble in fat, many of the potent sources are found and marketed dissolved in oil.

Chemists are well aware of the unstable nature of many oils, especially of the highly unsaturated oils, but it is surprising how few physicians and pharmacists take cognizance of the easy destructibility of these compounds. All vegetable and animal oils undergo an oxidative change in which oxygen is added as the double bond forming peroxides. The reaction is facilitated by heat, light, and contact with air; in addition, it is hastened by the presence of small amounts of previously oxidized oil. Chemically an oil may show appreciable amounts of peroxide before rancidity can be detected by taste or odor.

It was brought to our attention recently that a number of children under the medical care of an institution in Philadelphia were suffering from gastrointestinal disturbances apparently due to the cod liver oil given them. Upon investigation it was found that the oil dispensed to these families had deteriorated. This observation led us to investigate the condition of the oil being distributed by a large number of institutions in the city. Samples were requested of hospitals, public health centers, well baby clinics, and other child care organizations. These samples as obtained from the institutions in question were fairly representative of the oil being given to patients.

These oils were immediately brought to the laboratory, and the peroxide value determined.* The results of the preliminary survey indicated that there was a surprising uniformity of ignorance concerning the perishability of cod liver oil, on the part of those in charge of

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*Peroxide value is the number of milliequivalents of peroxide oxygen formed at the double bonds of unsaturated fatty acids during the development of oxidative rancidity. The absolute value varies with different fats; cod liver oil with a peroxide value of 10 is slightly rancid, and the rancidity is apparent to taste, and to smell when the peroxide value reaches 15.

dispensing comparatively large volumes of it. Few of the institutions took any precautions at all against the deterioration of a product relatively unstable in nature. It was the usual thing, in the smaller institutions, to find the main supply of cod liver oil on a shelf in the drug room, kept under the same conditions as such substances as boric acid or sodium bicarbonate. It was therefore more or less a matter of chance whether a sample was obtained soon after the original container was opened, or one from the bottom of the bottle.

In one institution, a hospital with a large pediatric out-patient department, the pharmacist had a special shelf built over the radiator in front of the window, where a ten-gallon clear glass bottle of cod liver oil was kept for handy dispensing. The bottle was arranged with a spigot at the bottom, so that the air bubbled up through the entire volume of oil whenever the spigot was opened. Better conditions could hardly be arranged for the rapid development of rancidity than these—heat, light, and aeration. And yet this system had been devised by a graduate pharmacist and had certainly been seen by many of the physicians of the hospital—an excellent hospital in a large city.

The institutions using very large volumes of cod liver oil were no exception to the general rule. In these were found large drums of cod liver oil kept in some convenient, seldom refrigerated, location; a hole in the top of the drum permitted equalization of pressure. In no institutions were any precautions taken to prevent contact of the air with the surface of the oil in the drum. However, in only the one hospital noted above was air actually allowed to bubble in through the spigot.

The spigot itself was found to be a cause of deterioration in the oils. All spigots seen were of essentially the same construction. A thin film of oil remained in and around the outlet after each using. This film dried and oxidized and acted as a catalyst for the further oxidation of the oil in the drum and of all subsequent samples passing through the spigot. Since the development of rancidity is an autocatalytic reaction, contact with this dried film was particularly serious in hastening the deterioration of the remaining oil.

A few exceptions to the general rule of conditions under which cod liver oil was kept should be noted. One institution bought 50 gallon drums and bottled it all immediately in small 6 ounce bottles. Several institutions bought oil in 5 and 10 gallon bottles and transferred it immediately to small containers. These samples were found to be in good condition. No other type of precaution against rancidity except transferral to small containers was found in any institution visited.

A summary of the preliminary survey is given in Table I.

As a result of this investigation of the chemical condition of oil being dispensed by institutions, it was decided to examine the cod liver

TABLE I

SURVEY OF COD LIVER OIL DISPENSED BY INSTITUTIONS
(17 REPRESENTATIVE SAMPLES SHOWN OUT OF TOTAL OF 42)

SAMPLE NO.	BRAND	WHERE OBTAINED	SIZE OF CONTAINER	WHERE KEPT	TEMP. COND.	HOW LONG IN CONTAINER	MEQ. PEROXIDE PER 1,000 GM. OIL	REMARKS
1.	C	Health center	5 gal.	Clinic room	Room temp.	Few days	5.0	Bottled immediately.
3.	A	General hospital	5 gal.	Drug room	Room temp.	3 days	6.8	
4.	B	General hospital	5 gal.	Cellar	Cool	3 mo.	10.7	
6.	D	General hospital	10 gal.	Over radiator	Warm	2 mo.	20.0	Aerated with each sample.
7.	D	General hospital	1 gal.	Drug room	Room temp.	1 mo.	24.0	
9.	D	General hospital	30 gal.	Cellar	Cool	8 mo.	34.7	Leaky spigot.
12.	C	General hospital	5 gal.	Drug room	Warm	3 mo.	24.9	
13.	B	Children's home	1 gal.	Drug room	Room temp.	1 mo.	7.8	
14.	B	General hospital	5 gal.	Drug room	Warm	3 mo.	24.9	
16.	B	Wholesale drug house	50 gal.	Drug room	Room temp.	1-2 hr.	2.0	Bottled and sealed immediately.
17.	B	General hospital	10 gal.	Drug room	Room temp.	2-3 mo.	7.0	
18.	B	General hospital	20 gal.	Drug room	Room temp.	3 mo.	6.4	
19.	C	General hospital	5 gal.	Near radiator	Warm	1 mo.	13.4	
20.	C	General hospital	5 gal.	Drug room	Room temp.	6-8 mo.	44.2	Accumulated samples poured into large container.
22.	C	Pediatric hospital	30 gal.	Cellar	Cool	2-3 mo.	22.0	Leaky spigot.
28.	B	General hospital	1 gal.	Drug room	Room temp.	1 mo.	18.7	
42.	B	Pediatric hospital	30 gal.	Cellar	Cool	4-5 mo.	48.0	Same as No. 22 2½ mo. later.

oil in the home of families using it. A second survey was therefore undertaken, in which were requested samples of cod liver oil representing "the last dose in the bottle." These samples were obtained from households of various economic levels and of different geographic areas in the city.

The results of this survey indicated that there was great variability in the condition of the oil when the bottom of the bottle was reached.

The degree of deterioration in this oil depended upon two factors, from the point of view of these surveys:

1. The quality of the original product obtained by the family.

2. The manner in which the family used the product once it was in their hands.

From the results of the survey of institutions dispensing cod liver oil, it can readily be seen that on the whole the quality of oils so obtained was relatively low. On the other hand, all the cod liver oil examined fresh from the original containers distributed by the reputable manufacturers was in uniformly good condition.

Families, like institutions, varied in their awareness of the perishability of cod liver oil. Unless mothers were told by their medical advisers that cod liver oil needed the same care they gave butter, the general tendency was to treat it as a "nasty smelly stuff" to be kept away from the rest of the family's food. As a result, the cod liver oil bottle was found isolated on the kitchen shelf or in the bathroom cabinet with other medicines. Another thing that militated against the more careful handling of this product was the economy of buying cod liver oil in large bottles of 16 to 32 ounces. These large bottles do not fit readily into the usually cramped ice box of most families.

TABLE II

CONDITION OF "LAST DOSE IN THE BOTTLE" OF COD LIVER OILS FROM HOUSEHOLDS (11 REPRESENTATIVE SAMPLES SHOWN OUT OF TOTAL OF 45)

SAMPLE NO.	BRAND	CONDITION WHEN OBTAINED	SIZE OF CONTAINER	WHERE KEPT	HOW LONG KEPT	CONDITION "LAST DOSE"
2	C	Excellent	32 oz.	Kitchen shelf	3 mo.	Very rancid
10	D	Excellent	12 oz	Ice box	1 mo.	Excellent
25	C	Excellent	8 oz.	Ice box	1 mo.	Excellent
26	B	Fair	8 oz.	Ice box	1 year	Moderately rancid
30	C	Fair	8 oz.	Ice box	2 mo.	Moderately rancid
31	C	Poor	8 oz.	Ice box	6 wk.	Very rancid
32	C	Poor	8 oz.	Ice box	1 mo.	Very rancid
35	C	Excellent	8 oz.	Ice box	1 mo.	Excellent
38	D	Excellent	6 oz.	Ice box	6 wk.	Excellent
39	D	Excellent	16 oz.	Bathroom	3 mo.	Very rancid
45	C	Excellent	32 oz.	Kitchen shelf	4 mo.	Very rancid

In this survey there was a high correlation between the method of handling cod liver oil and the awareness of the family doctor of its perishability.

The fact that quite a number of samples of cod liver oil representing "the last dose in the bottle" were found in as good condition as when the bottle was opened was convincing proof that the observance

of a few simple precautions could entirely eliminate the difficulty of feeding infants and children rancid cod liver oil. These simple precautions for the individual family might be listed as follows:

1. Obtain a standard product in good condition.

2. Keep it under refrigeration after the bottle has been opened.

3. Wipe off the mouth of the bottle after each using, to prevent the accumulation of rancid oil.

4. Obtain a supply sufficiently small to be consumed in a period of six weeks or less.

For institutions the problem is not quite so simple, since large scale purchases are an economy not to be ignored. However, as soon as the

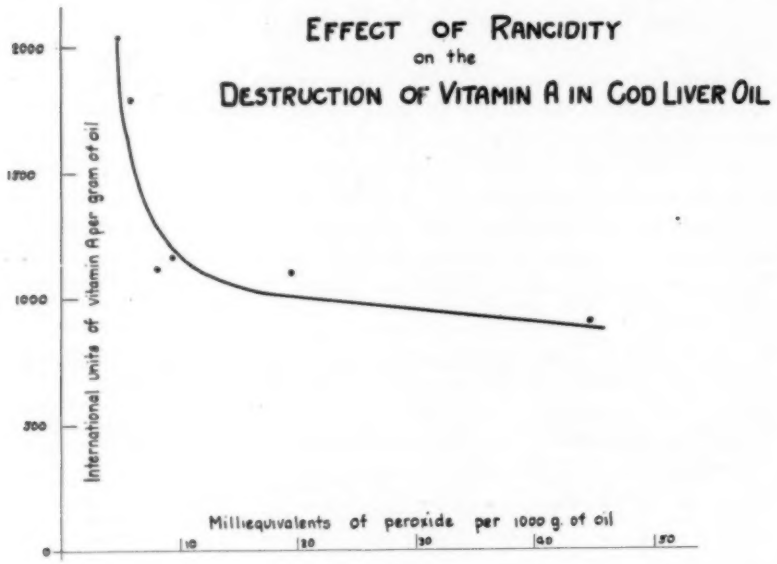

Chart 1.

medical groups responsible for the purchase and distribution of cod liver oil are awakened to the need of treating cod liver oil as a perishable food instead of as so much salt solution, half the problem, if not more, will be solved.

The question might be asked, "Why not feed children cod liver oil of this type; what harm, if any, does it do?"

The answer to this point has several phases:

1. Rancidity destroys vitamin A; the vitamin is easily inactivated by the peroxides of rancid oil. Dr. Charles E. Bills very kindly determined by the vitameter the vitamin A values on a series of cod liver

oils in various stages of deterioration. A correlation between the peroxide value of an oil and its vitamin A content was evident (Chart 1).*

2. The developing rancidity destroys the unsaturated linkages in fatty acids. Since linoleic acid is readily destroyed by the peroxides formed during the development of rancidity, rancid oil cannot be relied upon as a source of the fatty acids essential for nutrition. This is a more important factor concerning other oils, because cod liver oil is a poor source of linoleic acid even when fresh. It has been demonstrated previously in this laboratory[3, 4] that slightly rancid lard will not support life in dogs when it is the only source of fat in an otherwise adequate ration. The animals die from a disease which has been

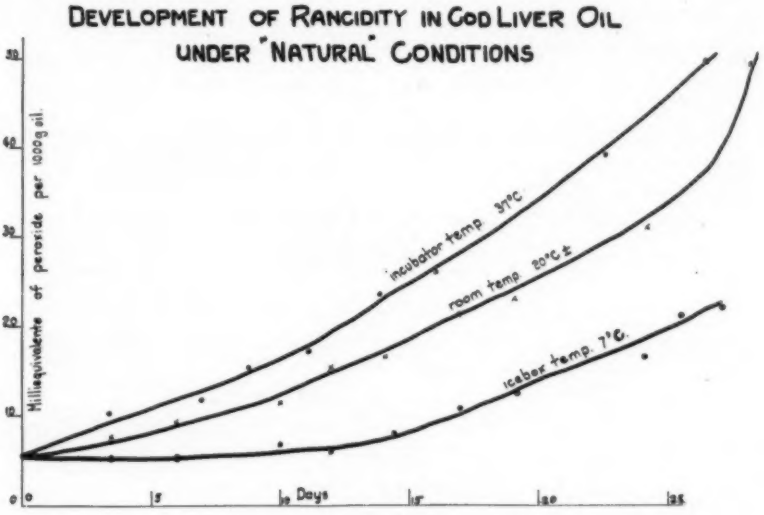

Chart 2.

termed "the oxidized fat syndrome," the etiology of which is probably a deficiency of the fatty acids essential for life.

3. It is generally believed that rancid fats are gastric irritants. The ingestion of even slightly rancid cod liver oil may well be the cause of the frequent digestive disturbances so often attributed by mothers to cod liver oil. An attempt is being made now to obtain statistical and clinical data on this point.

4. Rancid cod liver oil has a disagreeable taste and odor. At times a child's dislike of this food may be due to the fact that he has been offered a rancid oil.

*A report on the relation of rancidity to the destruction of vitamin A was presented before the American Oil Chemists' Society, Oct. 18, 1935, and will be published in their Journal in the near future.

Rancidity apparently has little, if any, effect on vitamin D. The prevention and treatment of rickets are usually the chief reasons for giving cod liver oil, and it is fortunate that this vitamin is practically unaffected by rancidity.

The keeping qualities of several samples of cod liver oil were investigated in the laboratory, under conditions designed to simulate various home conditions. It was found that a good grade of cod liver oil, kept in the ice box, would maintain its vitamin A potency and have a low peroxide value at the end of six weeks, but that the same oil, kept on an open shelf in the laboratory, exposed to ordinary daylight and the average fluctuations in room temperature, showed evident deterioration in a week and marked rancidity in two weeks. In addition, oil kept in an incubator under conditions simulating a warm kitchen or bathroom shelf became markedly rancid in three or four days, with a corresponding diminution in vitamin A potency (Chart 2).

SUMMARY

1. It was found that a large number of institutions dispensing cod liver oil failed to observe the precautions necessary to prevent the development of rancidity.

2. Many households likewise failed to observe the necessary simple precautions for preventing the development of rancidity in cod liver oil.

3. Only those families who obtained a good oil and kept it under proper conditions showed an oil in good condition at the end of the period of using a given supply.

4. Rancid cod liver oil has a disagreeable taste.

5. Rancid cod liver oil may be a factor in the digestive disturbances frequently reported by mothers after its use by their children.

6. Rancid cod liver oil is a poor source of vitamin A.

7. Rancidity in cod liver oil can be guarded against by observing the following simple precautions:

   a. Obtaining a product in good condition.
   b. Keeping it under refrigeration after the bottle has been opened.
   c. Keeping the mouth of the bottle free from dried residue.
   d. Obtaining a supply sufficiently small to be consumed in a period of six weeks or less.

8. Rancidity develops in other oils containing unsaturated bonds as readily as in cod liver oil.

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ABDOMINAL PUNCTURE IN THE DIAGNOSIS OF PERITONITIS IN CHILDHOOD

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ABDOMINAL puncture, I believe, has passed the experimental stage. Those who have used it would no more discard this procedure than would the neurologist eliminate spinal tap. So valuable a method to be of universal service should be safe and technically so simple that any physician with reasonable manual dexterity may apply it.

Fifteen years ago I proposed a method of abdominal puncture in infants and children.[1] It involved the use of a trocar, cannula, and glass capillary tube. Special precautions and special apparatus are necessary. At about the same time Neuhof and Cohen[2] were developing a different technic and they reported their results in 1926. I have used both methods extensively, and I have discarded the one I suggested.

In describing the technic, I shall refer to its use in infants and children only because my experience is confined to this age group. An infant's lumbar puncture needle, 2 inches long, 20 bore, is used. This type needle combines sharp point and obtuse bevel. I do not cocainize or incise through the skin although there is no objection to doing so. The needle with the stylet in place is inserted obliquely through the skin and then more perpendicularly with steady pressure until the sudden release or give indicates passage through the posterior sheaths of the abdominal muscles. A 2 c.c. or 5 c.c. syringe is attached, and gentle suction is applied. It is important to depress the syringe so that it lies flat on the abdomen, thus placing the point close to the peritoneal surface. There is a tendency for small quantities of fluid to form a film along the parietal peritoneum. Inasmuch as the minutest amount, a drop or part of a drop, may be diagnostic, the position of the needle best adapted to disclose such small quantities is an essential element in technic. As the needle leaves the abdominal cavity a small quantity of exudate may be aspirated. On several occasions changing the position of the needle, leaving it at rest for a few moments or puncturing in another part of the abdomen has been rewarded by the aspiration of a drop of fluid. Even when no fluid is to be seen in the syringe, forcible expression several times with the plunger may reveal a drop of pus. The usual site of puncture is below the umbilicus, avoiding the fixed viscera. The distended bladder should be evacuated. Where there is no fixation of the intestines puncturing the gut is a very remote possibility. Theoretically

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such an accident might happen. In many scores of punctures I have not seen it. The force needed to puncture freely movable gut is known to every surgeon; this may be confirmed at the autopsy table with the abdomen open by attempting to push a lumbar puncture needle through the intestine.

My experience has been confined to the problems of diagnosis of acute abdominal conditions occurring in pediatric practice and pediatric ward service and in comparison with that of the surgeon is limited. Only my own observations will be recorded, and therefore no attempt at completeness will be made. During these fifteen years I have not kept a record of all patients punctured, nor does the method lend itself to mere numerical analysis. I have chosen to report those cases which best illustrate the principles involved in the evaluation of abdominal tap and the therapeutic implications thereof. They do not represent a cross-section of my experience, which is much more favorable than these few cases suggest.

Negative punctures are obtained in all normal children. Although the abdominal contents are lubricated by fluid, for all practical purposes no appreciable amount exists. At any rate no one has ever collected or analyzed "normal" peritoneal fluid. In two noninflammatory conditions in infancy, marasmus and rickets, fluid at times has been obtained. The fluid is clear or mucoid and contains predominantly lymphoid and endothelial cells.[1]

Of more importance are the negative punctures in cases in which operation has revealed pus. I can recall this on only one occasion. Unquestionably, in a larger surgical group such cases are more frequent. Speculation as to the cause of these results is of less importance than the evaluation thereof. Where other evidence points to the diagnosis of peritonitis, a negative tap should not be permitted to modify the therapeutic procedure. To quote Neuhof and Cohen, "If in any given case the decision has been reached that operation is indicated, . . . operation should be proceeded with regardless of a negative puncture."

When a positive tap is obtained, the fluid should be examined grossly, spread on a slide, stained, and cultured. Examination of the smear is more vital than culture of the fluid because the decision as to therapy as a rule cannot await complete bacteriologic report. Indeed the smear may be of the greatest assistance even when apparently in conflict with the final report of culture. I am referring to those cases in which the examination of the smear shows pus without organisms, and culture reveals bacilli of the colon group alone or with gram-positive cocci. The following case illustrates this, as well as several common differential diagnostic problems: A boy, four years old, was admitted to the hospital with a history of upper respiratory infection, abdominal pain and vomiting. Examination showed a red eardrum, a red throat, and abdominal tenderness, at first diffuse and later somewhat more marked in

the right lower quadrant. The following possibilities were considered: (1) upper respiratory infection with abdominal symptoms, possibly due to mesenteric adenitis, (2) streptococcus peritonitis secondary to an upper respiratory infection, and (3) appendicitis. This decision is anything but academic, for therapy of these three conditions differs radically. Abdominal tap showed a single drop of pus; no organisms could be found. The child was operated upon at once; a gangrenous appendix was found; free fluid was present, and the wound was drained. Culture of the pus removed by tap showed colon bacillus and *Staphylococcus albus.*

In general, abdominal tap in cases of appendicitis will reveal one of the following: (1) a negative tap; (2) a positive tap without any organisms on smear and on culture either no growth or a pure culture of colon bacilli or colon bacilli with a coccus; or (3) a pure culture of streptococcus, a rare finding. Mandlebaum quoted by Neuhof and Cohen found streptococcus alone in only two of two hundred bacteriologic examinations. Nevertheless, it happens often enough to made certain equivocal clinical conditions particularly difficult to analyze.

The following case is illustrative of such a diagnostic difficulty and of some of the pitfalls in the evaluation of the results of puncture. A girl, five years old, was seen about thirty hours after the onset of abdominal pain which had been more or less continuous. Vomiting had occurred only once and then immediately after the administration of castor oil. A few days prior to this illness the child had an upper respiratory infection. Examination showed a severe follicular tonsillitis, râles at the right base, and the signs of a diffuse peritonitis. An abdominal tap produced a bare drop of pus demonstrable only on its expression from the needle and enough only for one good smear. There was doubt as to whether the gram-positive particles on the smear were artifacts or diplococci. The final decision was that they were probably cocci. As a rule in streptococcus peritonitis, fluid is more readily obtained, and organisms are numerous and easily demonstrated. Also, as mentioned above, streptococci not associated with the colon bacillus is an unusual finding in appendiceal peritonitis. Nevertheless, it seemed unwise on first examination to exclude the possibility of streptococcus peritonitis merely on the basis of finding a few rather than many cocci. The child was observed for eight to ten hours, during which time an intravenous drip was given. Tenderness became localized to the right lower quadrant, and a second tap was done which was negative. The child was operated upon, and appendicitis was found with a small perforation and localized peritonitis.

This case also confirms the previous experience that the absence of large numbers of cocci militates against the diagnosis of streptococcus peritonitis. One hesitates to use the words always and never. However, should the presence of many cocci and the absence of other organ-

isms prove to be a constant finding in streptococcus peritonitis, an important diagnostic criterion will have been established for a condition otherwise not easy to diagnose.*

The type of primary focus responsible for peritonitic exudate governs the type of peritonitis and modifies the evaluation of abdominal tap and subsequent procedure. Thus peritonitis following a thrombophlebitis may have a different course and will require different treatment from the peritonitis secondary to a pharyngeal angina, in which the organisms have entered the blood stream through the injured mucous membrane. The following case illustrates this principle: A four-year-old child was operated on for suppurative cervical adenitis. Within two days septic temperatures occurred and shortly thereafter signs of peritonitis. When I saw the child, there were, in addition to the neck wound, the usual signs of diffuse peritonitis. Abdominal tap showed pus with no organisms either on smear or culture. Blood transfusions were given, and subsequently with the help of the Queckenstedt procedure a diagnosis was made of thrombosis of the jugular vein draining the glands of the neck. Excision of the vein resulted in eliminating the primary focus, and the peritonitis subsided.

Pneumococcus peritonitis, particularly the type occurring in girls,[3] though radically different in pathogenesis from peritonitis of streptococcus origin, presents similar therapeutic and diagnostic problems. Space and time may be conserved by the statement, essentially accurate, that the paragraphs to follow on streptococcus peritonitis may be applied to pneumococcus peritonitis also.

In the diagnosis of streptococcus peritonitis in children abdominal puncture finds its greatest field of usefulness and exposes the most interesting diagnostic problems: the differentiation of abdominal distention secondary to pneumonitis from peritonitis and of streptococcus peritonitis from that secondary to appendicitis.

A girl, three years old, developed an upper respiratory infection six days previously and, on the day before admission, cough and vomiting. Respirations were rapid with dilating alae nasi, and the abdomen was distended. The chest showed impaired resonance at the right base and distant breath sounds. The case was considered to be pneumonia with abdominal distention. X-ray picture the following morning showed shadows on both sides, suggesting a moderate degree of infiltration and confirmed the diagnosis of pneumonia. Vomiting recurred, and thirty-six hours after admission the Hippocratic facies and marked abdominal fullness indicated something more than toxic distention. Pneumonia had progressed, and now there were obvious signs of it in both chests. Abdominal tap showed gram-positive cocci in chains. Despite the usual

*In a personal communication Dr. Neuhof informed me that in all his cases of streptococcus peritonitis, large numbers of cocci in chains were found.

intravenous therapy, the child died. Autopsy showed bilateral bronchopneumonia, purulent fluid in both pleural cavities, and diffuse peritonitis with large masses of fibrin.

The sequence of events probably was an upper respiratory infection with concomitant infection of lungs and peritoneum, or peritonitis preceded the pneumonitis. Critical review of this protocol indicates that in certain cases one cannot be satisfied with clinical and x-ray signs of pneumonia as sufficient explanation for abdominal distention. This child was probably too ill to be helped. My impression, however, is that the use of abdominal puncture in all cases of abdominal distention the cause of which is not obvious will reveal a larger number of cases of streptococcus peritonitis. Their early recognition, I believe, will lead to a lower mortality.

A boy, aged one and one-half years, was taken ill with an upper respiratory infection. In a few days the temperature rose, accompanied by cough, dilating alae nasi, vomiting and slight abdominal distention. When I saw the child, the problem consisted in the decision as to whether the distention was due to pneumonia or to diffuse peritonitis. The child was referred to Mount Sinai Hospital where roentgenograms taken at night on admission showed normal lung fields. The following morning there was a suggestion of localized abdominal tenderness. Thus the diagnostic problem again shifted and appendicitis had to be ruled out as the cause of peritoneal symptoms. Tap of the abdomen showed cocci in chains, and blood culture was positive for streptococci. The only therapy was a single blood transfusion and continuous intravenous saline. After seven days the continuous intravenous drip was discontinued, and the temperature came down to normal nine days after admission to the hospital.

This case illustrates the old triad of diseases causing similar and confusing clinical pictures—pneumonia, appendicitis, and streptococcus peritonitis. It indicates also the extent to which conservative therapy depends upon diagnostic puncture. This will be discussed again.

The third case is that of a child, aged six years, who one week previously had had a temperature of 105° F., which subsided in forty-eight hours. When I saw the child, she had a violently red throat and pharynx and a high fever. The following day there was vague abdominal pain and tenderness and in another twenty-four hours, vomiting, severe abdominal tenderness, and distention. After a negative x-ray picture of the chest abdominal tap revealed cocci in chains. Intravenous drip was started at once. In another two days the abdomen had become markedly distended, and respiration was embarrassed. Doctor Neuhof was asked to see the child. He advised operation to relieve the child of the insuperable burden of the accumulated abdominal fluid. I wish to emphasize first that operation was performed not primarily to drain the abdominal cavity, but to relieve respiratory embarrassment and to re-

duce the degree of toxic absorption by the withdrawal of whatever fluid would exude; and, second, that the operation was not an exploratory laparotomy in the usual sense of the term. Incisions were made in both flanks and large tubes inserted. This procedure might well be called a stab laparotomy to distinguish it from the usual operation for peritoneal drainage. From one side fluid gushed out in quantity and continued to drain for a period of weeks. From the other side only a small amount of pus flowed, and this ceased draining in about a week. Pneumonitis, empyema, and an infection of the kidney followed and were treated according to the respective indications. Transfusions and infusion of physiologic saline were the main forms of supportive therapy during the weeks of hospitalization. Complete cure took place.

Results of abdominal tap do not lend themselves to tabulation as do some laboratory tests. One cannot regularly associate bacteriologic reports with individual diagnoses. Yet there are some correlations so striking that their occurrence is almost pathognomonic. The finding of large numbers of gram-positive cocci in chains practically excludes appendicitis and fixes the diagnosis as either pneumococcus or streptococcus peritonitis, probably the latter. I have been interested in observing my own reactions and those of my colleagues in cases in which abdominal tap has cleared up the confusion in differentiating between pneumonia, appendicitis, and streptococcus peritonitis. The symptoms after the tap rearrange themselves around the new and central fact, pus-containing organisms in chains; new evaluations and different emphases are placed on observations, and reorientation of therapy takes place. One may dispute with one's colleagues concerning rebound tenderness, the significance of absence of vomiting, etc., but one cannot take issue with a drop of pus containing great numbers of cocci. That is why at times abdominal tap is one of the most dramatic and satisfying diagnostic events in clinical experience.

In emphasizing the importance of abdominal puncture in the diagnosis of streptococcus peritonitis, I do not mean to imply that the diagnosis cannot be made by other and older methods, for instance, exploratory laparotomy. However, assuming as more and more surgeons insist, that postponement of operation or complete avoidance thereof is the method of choice in pneumococcus and streptococcus peritonitis, one can proceed confidently with conservative therapy only if the conviction of correct diagnosis is backed by finding cocci alone in smears of fluid obtained by abdominal tap. In the first vital days of nonoperative treatment of a case of peritonitis supposedly of streptococcus or pneumococcus origin, no matter how confident one may be on clinical grounds of the correctness of the diagnosis, the haunting fear of appendiceal peritonitis might well shake the nerves of the most experienced clinician. The mere diagnosis of streptococcus peritonitis alone would be a sufficient apologia for abdominal puncture.

As stated earlier, the observations I have recorded make no pretense at completeness. The peritoneal complications of abdominal trauma, ruptured viscera, and inflammatory diseases other than that of the appendix have been excluded from my experience. On the other hand, this lack of completeness may be compensated for by the intimacy of the record. Gradually over the years it has been woven into the fabric of my clinical experience. Fifteen years ago I became interested in an objective method for the diagnosis of peritonitis because the paucity of signs and symptoms in this condition in infancy presented insuperable difficulties to diagnosis. At that time the possibility of adequate therapy was remote and most physicians were reconciled to a high mortality. Now, thanks to the changed viewpoint in therapy, particularly the use of transfusion and continuous intravenous drip and the postponement or avoidance of the usual exploratory laparotomy, the mortality among children has been and, I believe, will continue to be reduced. The prerequisite to this improved therapy is early diagnosis, and that in turn depends on abdominal puncture and experience in its evaluation.

Recent textbooks and reviews make no reference to this method of diagnosis. I doubt whether this is due altogether to the infrequency of reports to the medical profession in the past fifteen years by Dr. Neuhof and by me. Judging by the reaction of some of our surgical colleagues in medical meetings where abdominal puncture has been discussed, I believe that the neglect of this method is due to the assumption of technical difficulties and to the fear of puncturing the intestines. At the risk of needless repetition, I must emphasize again the simplicity and safety of the procedure; indeed, it is far safer than paracentesis of the pleural cavities.

CONCLUSIONS

1. Abdominal puncture is preeminently a safe procedure.

2. Negative tap should never be permitted to exclude the possibility of fluid in the abdomen or alter indications based on other clinical evidence.

3. Abdominal tap is of particular importance in differentiating the various types of peritonitis in childhood.

4. Reduction of mortality in streptococcus and pneumococcus peritonitis depends upon early nonoperative diagnosis, for which abdominal tap is indispensable.

5. Abdominal tap deserves to be incorporated among the usual diagnostic procedures.

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INDICATIONS FOR UROLOGIC INVESTIGATION IN CHILDREN

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INVESTIGATIONS in children have revealed that, with the exception of neoplasia of the lower urinary tract, adults and children are subject to the same urologic diseases. Moreover, urologic disease is not uncommon in the young, and this is extremely important: it is concerned chiefly with the pathology of anomalous development of the urinary tract. In most instances a rather complete examination is necessary to establish the correct anatomic diagnosis. The more common indications for urologic examination in infants and children in the order of their incidence are: chronic pyuria, disturbances of urination, pain or tumor along the urinary tract, hematuria not due to acute nephritis, and persistent acute urinary infection. Various phases of these indications are here discussed. Indiscriminate urologic instrumentation identifies an inadequate conception of the problem at hand.

PYURIA

Chronic Urinary Infection.—The majority of acute urinary infections are self-limited and clear up spontaneously irrespective of the treatment employed. When properly collected specimens of urine continue to show infection (always bacteria, usually pus also) despite a month of intensive medical therapy, the cause should be investigated. In females —irrespective of age—only specimens aseptically collected by catheterization are worth examination when urinary infection is under investigation or treatment. In males a satisfactory specimen may usually be obtained if, after retracting the prepuce and washing the glans and meatus with an antiseptic solution such as oxycyanide or bichloride of mercury 1:500, a few cubic centimeters of urine are voided before some is passed into a sterile receptacle. Only by such careful precautions can extraneous bacterial and cellular contamination be avoided. We may consider abnormal more than 6 pus cells per low power field of an uncentrifuged urine specimen. Absence of clumping of the pus cells is of no moment.

During the month of intensive "precystoscopic" medical therapy above referred to, focal infections should be eradicated as far as possible; intestinal elimination and nutrition should be improved; and a urinary

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antiseptic should be liberally administered. Methenamine in doses far larger than customarily employed and in conjunction with marked urinary acidulation (ammonium chloride, ammonium nitrate, or calcium chloride) remains the antiseptic of choice. In children the basis of dosage is 10 to 15 grains per day per year of age. Thus, a five-year-old child receives 50 to 75 grains of methenamine in twenty-four hours. Sufficient acidulant (I prefer ammonium chloride) is given to produce a urinary acidity of pH 5.5 or greater. As a rule the initial dose of the acidulant is the same as that of the methenamine, to be increased sufficient to make the urine turn methyl orange paper red (pH 5.5). Surprisingly large amounts of acidulant are sometimes required; the urine of a seven-year-old boy with a chronic staphylococcal and colon bacillary infection failed to become acid even to litmus (pH 7.0) with less than 200 grains (13.3 gm.) of ammonium chloride per day. The combined medication is given for four or five days. There is then a rest period of two or three days, and, if the infection is not eradicated as evidenced by two sterile cultures of catheterized specimens, the medication is resumed, in larger doses if possible. Vesical distress or hematuria due to methenamine therapy merely indicates that a high concentration of formaldehyde is being obtained in the bladder urine and is a favorable rather than unfavorable reaction. Yet the increasing severity of these symptoms may require reduction of the medication or its temporary discon-

TABLE I

PYELOGRAPHIC FINDINGS IN 580 CHILDREN WITH CHRONIC URINARY INFECTION

	RIGHT	LEFT	BILATERAL	TOTAL
Normal pyelograms				201
Dilatation of one calyx	3	3		6
Hydronephrosis	57	35	36	128
Pyelonephritis	7	8	41	56
Pyonephrosis	13	9		22
Hemi-pyonephrosis (reduplicated kidney)	7	4		11
Renal tuberculosis	5	3	1	9
Renal stone	6	2		8
Renal tumor	2	3		5
Renal ptosis	6	1		7
Renal rotation	1	1		2
Solitary kidney	5	2		7
Aplastic kidney	1	2		3
Ureter stricture	47	31	23	101
Dilated ureter	29	48	62	139
Ureter reduplication:				
Complete	16	9	8	33
Partial	9	13	3	25
Ureter kink	2	2		4
Ureter obstruction by aberrant vessel*	4	6	1	11
Ureter stone		2		2
Pyoureter	1	1		2
Ureter reflux (cystogram)	32	35	52	119
Ectopic orifice in urethra	2	3	1	6
Perinephritic abscess	1	2		3
Report or films lost, 14				

*Seven additional diagnosed; no confirmation.

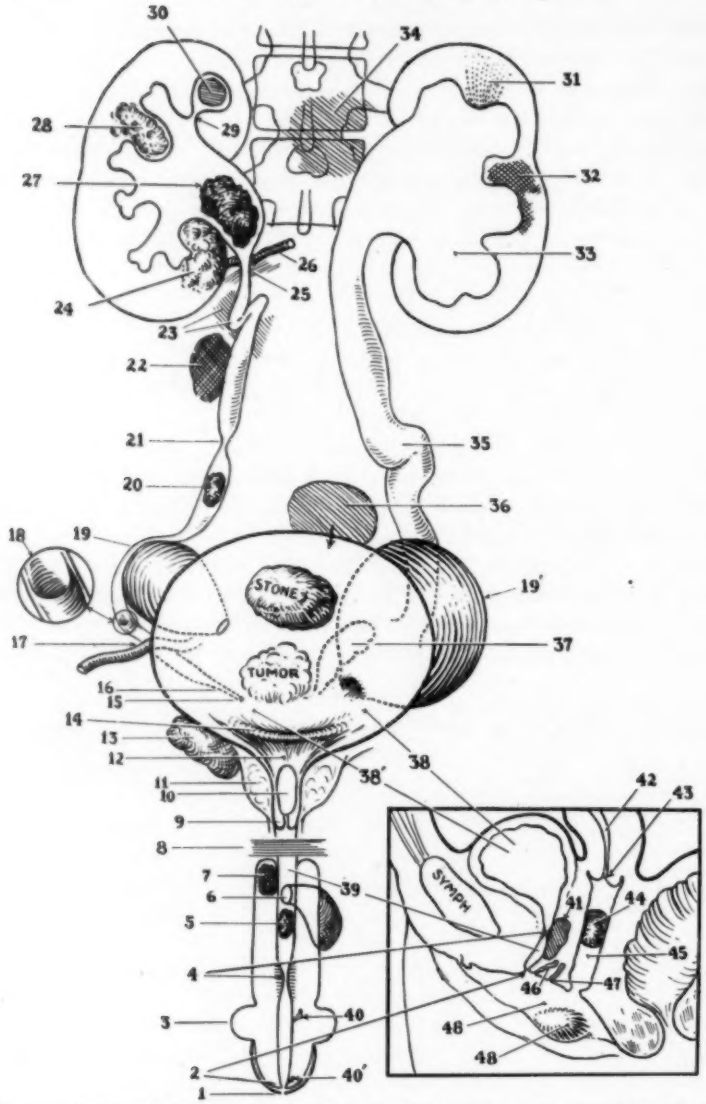

Fig. 1.—Direct and indirect causes of pyuria. These lesions, the clinical manifestations of which so commonly lead to the inadequate diagnosis pyelitis, either acute or chronic, can be identified only by a thorough urologic examination. *1*, Stenosis of prepuce; *2*, stenosis of urethral meatus; *3*, paraphimosis; *4*, urethral stricture; *5*, urethral stone; *6*, urethral diverticulum; *7*, periurethritis; periurethral abscess; *8*, cowperitis; chronic external sphincterospasm; *9*, congenital valves of posterior urethra; *10*, hypertrophy of verumontanum; verumontanitis; *11*, prostatitis; prostatic abscess; *12*, contracted bladder neck; median bar; *13*, periprostatitis or pelvic suppuration; *14*, mucosal fold at bladder outlet; trigonal curtain; *15*, stricture of ureteral meatus; ureterocele; *16*, ureterovesical junction stricture; *17*, vascular obstruction of lower ureter; *18*, congenital ureteral values; *19*, ureteral obstruction by diverticulum compression; *19*,[1] diverticulum; *20*, ureteral stone; *21*, ureteral stricture; *22*, periureteritis; periureteral phlegmon or abscess; *23*, ureteral kink; periureteral fibrous bands; *24*, renal tumor; *25*, ureteropelvic junction stricture; *26*, aberrant vessel obstruction of

tinuance. If the urine cannot be sterilized during two to three weeks of this treatment, further similar efforts are likely to be fruitless and the child should be examined urologically.

When methenamine therapy alone cures urinary infection, one is almost always safe in assuming that no important urinary obstruction exists. On the other hand, the ketogenic dietary treatment of urinary infection is about 60 per cent successful and often in the presence of severe urinary anomalies and marked obstruction. Yet in these cases, the fact that chronic pyuria existed and required the ketogenic diet for its eradication, entitles the child at least to an excretory urographic examination to determine what factors (especially obstructive) helped to perpetuate the infection.

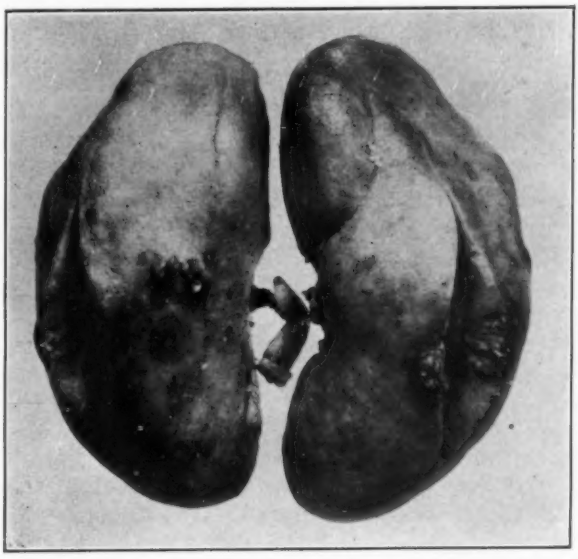

Fig. 2.—Multiple left renal carbuncles in a nine-year-old girl with "persistent acute pyelitis." The findings on urologic investigation indicated the renal exploration. Nephrectomy.

Among the more common causes of chronic pyuria are obstruction in the urethra, at the vesical outlet, or in the ureter, urinary stone, tumor, tuberculosis, vesical diverticulum and neuromuscular disease of the urinary tract (Fig. 1). The upper tract lesions demonstrated by pyelography in 580 instances of chronic pyuria in children personally examined are shown in Table I.

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upper ureter; *27*, pelvic stone; *28*, renal tuberculosis; *29*, stricture of calyceal outlet; *30*, calyceal stone; *31*, pyelonephritis; *32*, pyonephrosis; *33*, "pyelitis"; infected hydronephrosis; *34*, perirenal suppuration invading urinary tract; spinal disease (Pott's, etc.); *35*, hydroureter; *36*, pericystic abscess rupturing into bladder; *37*, seminal vesiculitis; *38*, neuromuscular vesical disease; *38*,[1] cystitis; *39*, urethritis; *40*, folliculitis (Littre); *40*,[1] folliculitis (Morgagni); *41*, periurethritis; periurethral abscess; *42*, endometritis; *43*, cervicitis; *44*, foreign body in vagina; *45*, vaginitis; *46*, skenitis; *47*, folliculitis of introitus; *48*, bartholinitis.

Persistent Acute Urinary Infection.—When acute urinary infection, usually designated as pyelitis, persists unabated for five days despite the usual treatment, a urologic examination is indicated. Infected hydronephrosis will most often be discovered although parenchymal suppuration may exist—locally as renal carbuncle or general, for example, as acute diffuse focal suppurative nephritis. An eight-week-old girl suffered for more than a week a continuous "acute pyelitis" with a temperature persistently around 104° F. Urologic examination revealed a congenital right ureteropelvic junction stricture with infected hydronephrosis (pure culture Flexner dysentery bacillus). The opposite side was normal. Passage of a ureteral catheter to the right pelvis coincident with the urologic examination effectively established renal drainage, and the temperature promptly fell to normal where it remained. On the other hand, in a nine-year-old girl alleged to have a persistent "acute pyelitis" and a typhoidlike temperature for four weeks, urologic examination indicated a left renal exploration which in turn revealed two moderate size renal carbuncles (Fig. 2). In a boy and in a girl of eight weeks, urologic examination instigated by persistent "acute pyelitis" disclosed renal carbuncle with a secondary perirenal abscess. Although the urologic attitude in renal infection should be one of conservatism, especially in children, when the high temperature of "acute pyelitis" shows no inclination to diminish in five days, ascertain the reason. Moreover, in known renal infection in a child, a persistent picket-fence temperature or repeated chills followed by drenching sweats calls for a complete urologic investigation. A renal or perirenal surgical suppurative lesion is almost certain to be found.

DISTURBANCES OF URINATION

Frequency and difficulty of voiding and incontinence are the commonest disturbances of urination in the young. Urinary frequency is usually due to inflammation at the bladder outlet or in the posterior urethra, even in the absence of infection. Yet pyuria and other evidence of infection usually accompany marked frequency. Frequency may also be caused (1) by obstruction along the lower urinary tract and with or without residual urine, (2) by vesical stone, or (3) by a reflex from a lesion of the kidney or ureter.

Urinary difficulty is caused by obstruction at or peripheral to the bladder outlet or by neuromuscular vesical dysfunction. Dysuria is most often produced by congenital contracture of the vesical outlet, cord bladder, congenital posterior urethral valves, congenital hypertrophy of the verumontanum, or urethral stricture, notably at the meatus.

Urinary incontinence may be true, as when due to vesical paralysis, ectopic ureteral orifice, or in certain types of epispadias, for example, and false or paradoxical, when present as an overflow in infravesical obstruction.

Incontinence in children, whether nocturnal (bed-wetting), diurnal (clothes-wetting) or both, usually accompanied by frequency, regularly leads to the diagnosis enuresis. Although it is likely that in 95 per cent of all enuretic children the condition is purely functional, those patients four years of age and older who are not cured in two or three months by medical, psychotherapy, or physiotherapy, should be subjected to urologic examination. In about 60 per cent of this treatment-resistant group an organic basis for the enuretic manifestations will be found. Most commonly encountered are congenital obstruction at the bladder outlet and in the urethra, neuromuscular disease of the bladder wall and outlet, urinary infection, masturbatory prostatitis in males and urethritis in females. One in seven of this group in my series had residual urine, i.e., they could not completely empty their bladders.

Only by a thorough urologic examination can any organic cause of enuresis be discovered. Curious lesions will sometimes be revealed: for example, in a six-year-old boy with enuresis, cystourethroscopy disclosed a pedunculated polyp springing from the roof of the posterior urethra immediately overlying the verumontanum. The tumor extended back into the bladder to produce not only irritation but a ball valve type of obstruction at the bladder outlet. Suprapubic excision of the growth cured the enuresis. In a 14-year-old boy with bed-wetting since birth three large horny polyps arising from the posterior end of the verumontanum caused the etiologic posterior urethral irritation. Cautery destruction of the growths cured the enuresis. In two instances the true cause of the enuretic manifestations—renal tuberculosis—was recognized only by urologic examination and required nephrectomy.

PAIN

Ureteral colic simulates enteric colic in the young, but, as a rule, in the former the pain is intermittent, and diarrhea is absent. A dull ache in the loin of a child is most likely to indicate hydronephrosis. Crying on urination always demands a urologic examination; deep urethral obstruction, severe urethrovesical infection, vesical calculus, renal tuberculosis, or ulcerated meatus are most likely to be found. It is important to note that urologic pain may exist even though the urinalysis is negative. For example, during the past year on the Children's Medical Division at Bellevue Hospital I examined two girls solely because of persistent left loin pain. In the first patient, five years old, the pain was caused by a fixed (congenital ?) left ureteral kink about 5 cm. below the ureteropelvic junction (Fig. 3). A transverse band of scar tissue firmly maintained the kink irrespective of the position of the patient—upright or Trendelenburg. The upper ureter was mobilized and the kidney suspended. The stricture at the site of the kink (evident in Fig. 3) has been treated by periodic instrumental dilatation. In a four-year-

old girl, a congenital diverticulum of the lower ureter—an extremely rare condition—was demonstrated and removed. In each instance the pain was relieved.

TUMOR

Congenital hydronephrosis is the commonest abdominal tumor in children; embryonal adenomyosarcoma of the kidney—the so-called Wilms

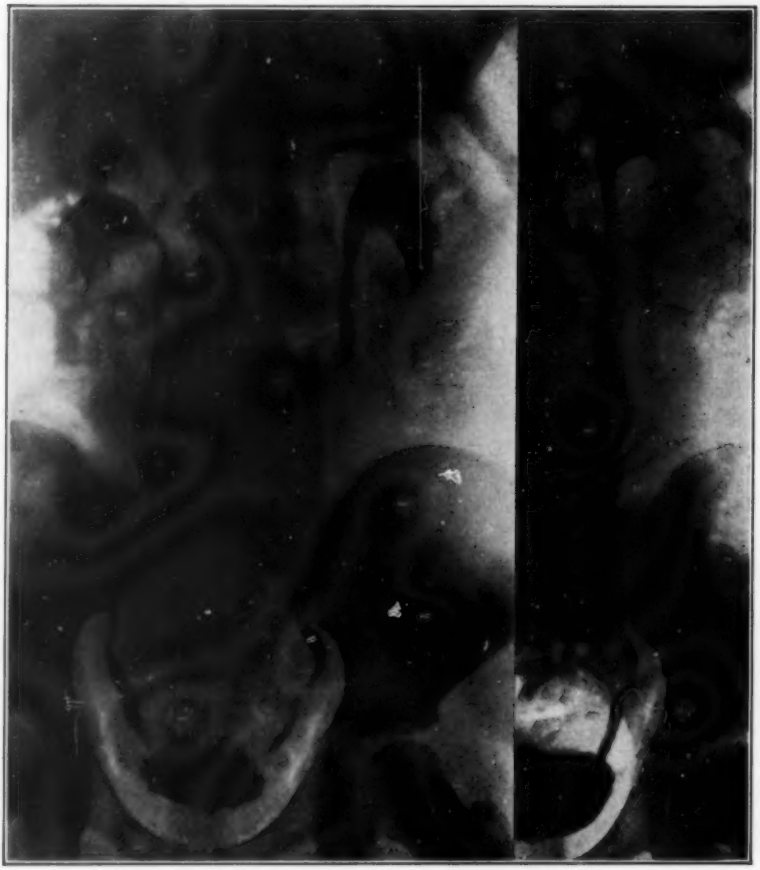

Fig. 3.—*A*, Left ureteral kink (congenital?) in a five-year-old girl in whom the sole indication for urologic examination was persistent pain in the left side. Urinalysis was negative. *B*, The upper urinary tract following straightening of the kink and nephropexy.

tumor—is the commonest malignant abdominal tumor in children. Yet the abdominal tumor may even be a mobile solitary kidney, as I have observed in a six-year-old girl. Neuroblastoma of the adrenal is nearly half as common as Wilms renal tumors in children, and the two condi-

tions can be differentiated only by urologic examination or surgical exploration. Most tumors erroneously diagnosed retroperitoneal sarcoma in children are neuroblastomas. The diagnosis of hydronephrosis, renal

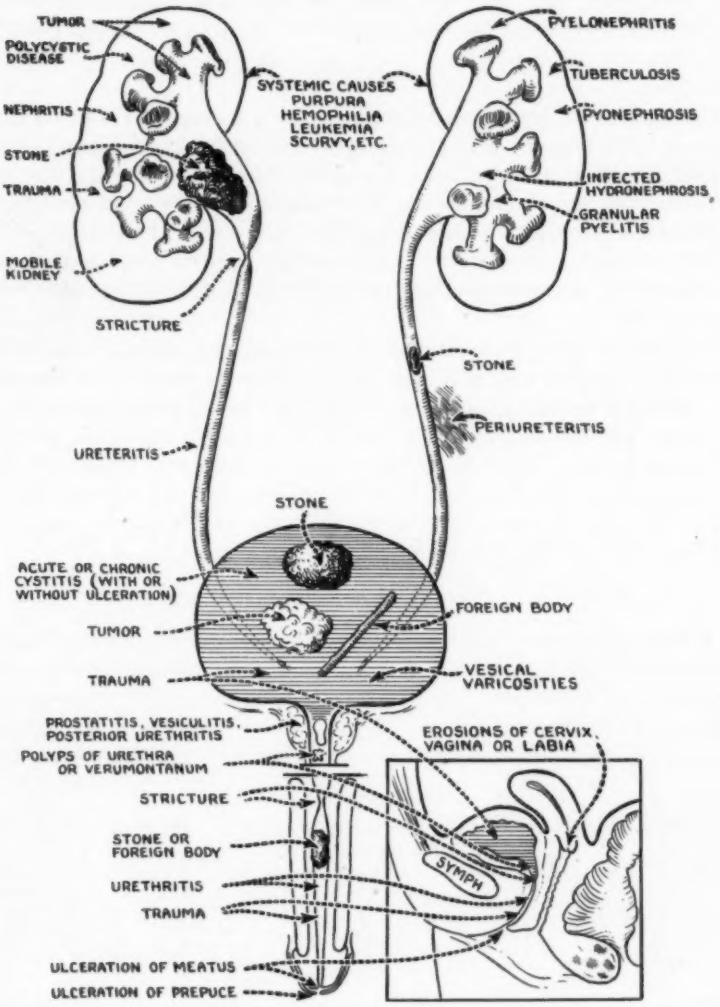

Fig. 4.—Causes of hematuria. Only by urologic examination, surgical exploration, or autopsy can most of these lesions be identified.

neoplasm, adrenal tumor, or perirenal abscess must usually be determined by ureteral catheterization and pyelography.

A suprapubic mass in a child is almost always a distended bladder; the latter is accurately identified by catheterization. The more common

causes of vesical distention are contracture of the bladder outlet, congenital valvular obstruction of the posterior urethra, and neuromuscular disease of the sphincterospastic type. On the other hand, in a twelve-year-old girl sent to the hospital because of a large suprapubic tumor, urologic examination proved the mass to be a fused ectopic pelvic kidney.

HEMATURIA

Hematuria not due to hemorrhagic nephritis demands a thorough urologic investigation as early as possible and preferably while there is bleeding. Although hematuria in a child should make one first suspect nephritis, then renal neoplasm or tuberculosis, the variety of conditions which may cause blood in the urine (Fig. 4) suggests the usual futility of attempting to make the anatomic diagnosis without resort to urologic examination. An ulcerated meatus is a frequent cause of hematuria in young boys.

Finally, in several children who presented the clinical picture of chronic interstitial nephritis, urologic investigation revealed an obstructive basis for the renal pathology. Although I would surely not advise a complete urologic study in every child in whom the diagnosis chronic nephritis is made, it is strongly urged that an excretory urographic examination be made. The data thus gleaned are obtained without risk to the child and, in some instances, will prove invaluable in directing further investigation or treatment.

CYSTOSCOPIC REACTIONS

Inhibited by fear of undesirable sequelae, most physicians continue to refuse their young patients the benefits of modern urologic studies when urologic symptoms exist. Irritation in the urethra and at the vesical outlet is to be expected for a few hours or even a day or two following cystoscopy. Yet this is surely no contraindication to urologic investiga-

TABLE II

POSTCYSTOSCOPIC FEBRILE REACTIONS IN CHILDREN: AUTHOR'S SERIES, 752 EXAMINATIONS

DAYS*	1	2	3	4	5	6	8	TOTAL
Less than 100.5° F.	42	11	2					55
100.6-103°	27	9	3	2	1	1	1	44
103.1-104.5°	5	2	2	2		1		13
Over 104.5°	1	1						2
None								648
Reactions 2°F. over	59							
Reaction incidence	7.9 per cent							

*Days before temperature returned to previous normal.

tion in either a child or adult. Reactions following observation cystoscopy in children are almost nil. I have performed such observations in over 600 children with enuresis and have not observed a reaction

more severe than a local urethral irritation, either in those patients kept in bed overnight or in those, by far the greater number, immediately discharged. Postinstrumental reactions in 752 consecutive complete urologic examinations in children have been studied and tabulated. A reaction—a temperature rise of 2° F. or over—may be expected in about one of every twelve (7.9 per cent in my series) children subjected to cystoscopic studies of the upper urinary tract. Yet three-fourths (77 per cent) of the children in my series with reactions were given a general anesthetic which doubtless explains many of the transient fevers. Comparison with a group of 300 adults similarly studied by Wehrbein on the urologic service of Bellevue Hospital shows that reactions occurred in the young less than half as often and were much less severe. For these reasons, the possibility of postinstrumental reaction as a sole contraindication or objection to urologic examination of an infant or child must be disregarded.

140 East Fifty-Fourth Street

Critical Review

PROGRESS OF PUBLIC HEALTH AS IT RELATES TO THE CHILD

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MORTALITY AND MORBIDITY

THE mortality statistics thus far available for the year 1935 in the United States present on the whole a favorable picture. Preliminary figures which will appear in Public Health Reports dated May 1, 1936, and are based upon reports from twenty-six states, have been made available in advance by the United States Public Health Service. Certain of those particularly related to child health are presented in Table I. This table shows a very gratifying further decline in total infant mortality over the preceding year and a new low of 52 per thousand live births for the area. Maternal mortality only shows a fractional decline as in each recent year. The specific rates show a decided decline over the previous year for measles, whooping cough, and diarrhea and enteritis under two years, with slight improvement in those for typhoid fever, diphtheria, tuberculosis and nephritis. The rates for scarlet fever, acute anterior poliomyelitis, meningococcus meningitis, influenza, pneumonia, and diseases of the heart were increased over those for 1934. In only scarlet fever and heart disease did these rates represent a new high for the five-year period.

Infant mortality, after a long period of decline, is apparently increasing in some places. Rates are especially high in certain cities. Little progress has been made in reducing maternal deaths or deaths of infants under one month of age. The need for a factual basis for special efforts to lower infant and maternal mortality in certain cities is indicated by the great variations in rates. The cities with the highest rates in 1934 lost between two and three times as many babies, proportionately, as the cities with the lowest rates. Similar variations are found in maternal mortality rates. Comprehensive studies of infant and maternal mortality in cities are greatly needed as a basis for devising the most effective programs for saving the lives of mothers and babies, as shown by previous studies made by the Children's Bureau and summarized in 1925 in *Causal Factors in Infant Mortality*.[1] It is understood that with this need in mind a new study of infant mortality and maternal care in cooperation with state health departments will be initiated by the Children's Bureau in the near future in an urban community with a population of 250,000 to 350,000.

Among the reportable diseases, meningococcus meningitis came into prominence during the year 1935 because of an unusually high and

[1]Woodbury, Robert Morse: Causal Factors in Infant Mortality, United States Children's Bureau Publication, No. 142, Washington, 1925.

progressively mounting incidence. The number of cases reported for the entire year was from two to three times the number reported in each of the preceding two years in every section of the country.[2] The year 1936 started with an incidence twice that at the same period of 1935 and three times that of 1934, indicating a continuing advance. Meningococcus meningitis tends to appear in cycles, with a few years of advancing rates followed by a number with progressively declining rates. The last peak year was 1929, and it now appears that 1934 was

TABLE I

SUMMARY OF MORTALITY FROM CERTAIN CAUSES IN A GROUP OF STATES, 1931-1935*†

DISEASES (NUMBERS IN PARENTHESES ARE FROM THE INTERNATIONAL LIST OF CAUSES OF DEATH, FOURTH REVISION, 1929)	1935	1934	1933	1932	1931
25 States (population July 1, 1935—83,358,000)	*Death Rate per 1,000 Population*				
All causes	10.8	10.9	10.6	10.7	11.0
24 States (live births 1935—1,299,048)	*Deaths Under 1 Year per 1,000 Live Births*				
Total infant mortality	52	58	56	56	60
18 States (live births 1935—1,021,952)					
All infant mortality except malformations and early infancy	21	24	23	24	28
24 States (live births 1935—1,299,048)	*Deaths of Mothers per 1,000 Live Births*				
Maternal mortality	5.3	5.5	5.6	5.9	6.2
26 States (population July 1, 1935—85,110,000)	*Death Rate per 100,000 Population*				
Typhoid fever (1, 2)	1.9	2.3	2.5	3.0	3.5
Measles (7)	2.7	4.3	1.7	1.4	2.4
Whooping cough (9)	3.4	5.1	3.1	3.9	3.6
Scarlet fever (8)	2.1	1.9	1.9	1.9	2.0
Diphtheria (10)	2.3	2.4	2.7	3.5	4.0
Acute anterior poliomyelitis (16)	0.8	0.6	0.6	0.7	2.0
Meningococcus meningitis (18)	2.0	0.8	1.1	1.3	2.1
Influenza (11)	19.2	15.0	22.8	25.2	23.3
Pneumonia, all forms (107-109)	80.1	78.7	69.0	75.7	80.1
Tuberculosis, all forms (23-32)	52.5	54.5	56.5	60.2	64.5
25 States (population July 1, 1935—83,358,000)					
Diarrhea and enteritis under 2 years (119)	7.6	10.7	9.4	9.4	13.1
25 States (population July 1, 1935—81,780,000)					
Nephritis, all forms (130-132)	82.2	84.6	81.8	85.3	85.0
24 States (population July 1, 1935—80,027,000)					
Diseases of the heart (90-95)	254.9	249.6	230.8	223.9	216.7

*The twenty-six states upon which most of these rates are based are California, Connecticut, District of Columbia, Georgia, Idaho, Illinois, Indiana, Iowa, Kansas, Louisiana, Maryland, Michigan, Minnesota, Mississippi, Montana, Nebraska, New Jersey, New York, North Carolina, Pennsylvania, South Carolina, South Dakota, Tennessee, Virginia, Wisconsin, Rhode Island. The state group used for all causes and diarrhea and enteritis omits South Carolina. The twenty-five-state group for nephritis omits North Carolina, but includes South Carolina not listed above. The twenty-four-state group for heart diseases omits North Carolina and South Carolina.

†Made available by the United States Public Health Service from data to appear in *Public Health Reports*, issue of May 1, 1936.

the low of the last cycle and that we are again in a period of advancing rates. The mortality rate for 1935, previously referred to, reflects this change.

[2]Public Health Reports, U. S. Public Health Service, Vol. 51. Monthly summaries of *Current Prevalence of Communicable Diseases in the United States.*

Scarlet fever was also on the increase in most sections during 1935, especially in the West, North Central, and the Mountain and Pacific regions. The trend during the early months of this year has also been continuing upward. The mortality rate, however, suggests that it has remained of a relatively mild form.

There was a sharp rise in the reported number of infantile paralysis cases for the country as a whole, the figures for the summer and fall months running three times seasonal expectancy; but this was due to exceptionally high incidence along the Atlantic seaboard. Starting in North Carolina and Virginia in July, this wave of the disease spread up through New England and the Middle Atlantic States, causing an incidence in some of these as high as eighteen times that for 1934. By January, 1936, however, the reported cases for the country as a whole had fallen to a low for the past eight years.

The incidence of smallpox for the country as a whole was also high because of a rise well above recent years in the Mountain and Pacific and North Central regions, but, exclusive of these states, the country showed no increase.

During the year 1934 measles had been unusually prevalent, especially in New England, the Middle Atlantic, and North Central sections. This high incidence persisted during the first half of 1935, but thereafter it began to fall, and by January, 1936, was relatively low for the season, very much below the 1935 level. Although diphtheria has continued to decline slowly in most sections, there was a quite marked increase in some sections during the summer season.

Exclusive of the reportable diseases, data on the health of the child population of the United States remain negligible. Several reports during the year by Collins,[3] based on records dealing with 9,000 families in eighteen states, subjects of an intensive survey carried on by periodic visits over a twelve-month period, throw light upon the general incidence of illness as well as the occurrence of certain specific diseases during the 1928-1931 period. These reports deal with the incidence, mortality, and estimated case fatality by age, by groups of disease, and by certain specified diseases, within a representative sample of the population. These reports seem of special interest because of the method which they suggest for obtaining from year to year a cross-section of the changing state of health of the nation. Such information should prove of considerable value as an indicator of the changing incidence or severity of particular diseases, of the success of preventive procedures, or of the adequacy of medical care.

Further reports by Perrott and Collins[4] and Palmer[5] tend to confirm the generalizations made in our review last year, in considering the effect of the depression on the health and growth of children. They produce further evidence from quite different approaches as to the relation of sickness to income and income change. They show especially that the highest illness rates and the most pronounced interference with normal progress during childhood occur among those families which have suffered the greatest change in standard of living. The newly poor are apparently unable to adjust satisfactorily, and they and their children show more pronounced effects than do those previously in the same income group.

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[3]Collins, Selwyn D.: Public Health Reports, U. S. Public Health Service, February 22, April 12, and October 11, 1935.

[4]Public Health Reports, U. S. Public Health Service, May 3, 1935.

[5]Public Health Reports, U. S. Public Health Service, August 16, 1935.

STILLBIRTHS AND NEONATAL MORTALITY

Much of the confusion regarding fetal mortality is recognized to be due to lack of uniformity in the use of terms and in the completeness of registration of stillbirths. For this reason the recommendations of the subcommittee on Stillbirths of the Committee on Accuracy of Certified Causes of Death of the American Public Health Association are deserving of attention.[6] The Committee recommends uniform state laws, to replace the wide variety of present requirements for reporting of stillbirths, and a standard certificate of stillbirth. It also makes recommendations as to the definition of stillbirth, live birth, early abortion, late abortion, and premature labor. As to a stillbirth, the recommendation is that "only such stillbirths as are estimated to represent products of conception of twenty weeks' duration or more—should be registered," but that "no child that shows any evidence of life after birth should be registered." Heart beat, respiration, or movement of voluntary muscle is to be accepted as evidence of life. Adoption of standard procedures in these matters, as well as in nomenclature of fetal and neonatal disease, should be expedited throughout the states.

Clifford[7] in his latest report from the Boston Lying-In Hospital indicates that neonatal mortality in that hospital has declined during the past ten years from 28 per 1,000 live births in 1925 to 20 in 1934. He shows that this decline has been due entirely to a decline in the premature infant death rate from 18 per 1,000 in 1925 to 10 in 1934. That is, the rate for mature neonates was 10 per 1,000 both in 1925 and 1934. The reduction in premature infant mortality obtained during the first two weeks of life at the Boston Lying-In Hospital has not occurred generally throughout the country, and it is apparently largely for this reason that neonatal mortality rates remain so constant and high. There is a growing realization that it is among premature babies that our communities may at the moment expect the greatest return from increased effort to conserve life. Premature infants represent only from 3 to 4 per cent of all births and are therefore a relatively small sample of the population upon which to expend intensive effort. From this small group, however, are drawn a major portion of all neonatal deaths and approximately one-third of the total infant mortality.

With this situation in mind Bundesen[8] has established a program in Chicago which should be watched with interest by other communities. With the assistance and advice of the medical profession in Chicago, the Department of Health has utilized its resources and exercised its authority in behalf of the prematurely born infant. Regulations have been adopted specifying the minimum accommodations and equipment and the minimum of nursing care which must be made available by any institution undertaking to care for a premature infant. Prompt reporting by telephone of all premature births is also required. Facilities are constantly available for the immediate transfer of those premature infants born in places not suitably provided to properly equipped nurseries. Arrangements are made for appropriate follow-up health service at special premature conferences for infants discharged from premature services. An educational program for medical, nursing, and lay groups has also been established. Realizing the importance of adequate nursing care for the successful protection of premature infants,

[6] Stillbirths. Am. Public Health Association Year Book, 1935-36, pp. 244-249.
[7] Clifford, Stewart: A Study of Neonatal Mortality, J. PEDIAT. 8: 367, 1936.

a course of training in the care of premature infants has been developed, and nurses so trained are being assigned by districts to specialize in the care of premature babies born within their districts.

The Chicago program has been in operation for too short a period to draw any definite conclusions as to the effect it will have upon infant mortality in that city. Recent figures,[9] however, show a premature infant mortality for the city of Chicago of 13.9 per 1,000 total live births in 1935 as compared with 15.6 for 1934. The new regulations regarding the reporting of premature births are apparently being taken seriously by physicians generally, for, of the 49,425 births in the city during 1935, 1,862 were reported as premature births; a rate of 3.76 per cent. This is as high a rate as would be expected in any completely reported series.

PUBLIC HEALTH SERVICES IN THE UNITED STATES

Because of the consideration given to public health conditions in foreign countries in our review last year and the lack of outstanding changes deserving comment at this time, attention here is confined to changes within the United States. The one outstanding change has been the reversal of the trend toward curtailment of all public health activities, due almost entirely to the passage of the federal Social Security Act. Recent reviews in this series have dealt with the adverse effects of financial conditions upon public health services, but in the last report the provisions of the Social Security Bill then before Congress were considered. This bill in much the same form as then outlined shortly thereafter became an act, but no funds were made available until February, 1936. These funds are now being put to work in various sections of the country. The grants for general public health are being administered by the United States Public Health Service. Those for maternal and child health and for crippled children are being administered by the Children's Bureau of the Department of Labor. The future course of public health as it relates to maternal and child health in this country will be profoundly affected by this act and the policies adopted by the Children's Bureau for its administration. Therefore, it has seemed wise to devote the remainder of this review to a consideration of the activities of the Children's Bureau and especially to the provisions of the act and the policies of the Bureau in regard to them. The plans of the various states as thus far adopted for the utilization of federal grants are also presented in brief outline. Much of the material which follows has not as yet been published. It has been made available to the reviewer by the assistant chief of the Children's Bureau, who is in direct charge of all health services.

THE CHILDREN'S BUREAU OF THE DEPARTMENT OF LABOR

The Children's Bureau of the United States Department of Labor has three divisions concerned with maternal and child health: (1) the Division of Research in Child Development, Ethel C. Dunham, M.D., director, set up under the organic act establishing the Children's Bureau (1912) and (2) and (3) the new divisions set up under the Social Security Act to administer grants-in-aid, the Maternal and Child Health

[9]Hess, Julius H.: A City-Wide Plan for the Reduction of Deaths Associated With and Due to Prematurity, J. PEDIAT. 8: 194, 1936.

[9]Personal communication from Julius H. Hess.

Division, Albert McCown, M.D., director, and the Crippled Children's Division, Robert C. Hood, M.D., director. All three of these divisions are under the immediate supervision of Martha M. Eliot, M.D., assistant chief of the Children's Bureau.

DIVISION OF RESEARCH IN CHILD DEVELOPMENT, 1935-1936

The following projects were being conducted by the Division of Research in Child Development during 1935 and the first half of 1936.

Infant Mortality in Memphis, Tenn.—The Children's Bureau was requested during the year to study causes of the high infant mortality rate in Memphis, which in 1932 exceeded that of all cities of 100,000 or more population in the United States and had increased from 1932 to 1934. The outstanding fact which emerged from the investigation made by one of the Children's Bureau physicians was that preventive health services for mothers and infants and services for the care of sick infants had not expanded in proportion to the needs of the past five years, which were increased chiefly by economic conditions and by incorporation within the city of a populous territory with high infant death rates and few resources. A number of recommendations were made, some of which have already been followed.

Conditions Affecting Child Health.—In cooperation with the National Organization for Public Health Nursing and fifty-seven public health nursing agencies in cities in thirty-three states, the Children's Bureau collected and tabulated information as to overcrowding, use of certain essential foods, and physical defects in children. The study covered more than 10,000 families, including some 54,000 individuals of whom 28,000 were under sixteen years of age. Overcrowding in the homes was shown by the fact that 57 per cent of the families reported more than two persons per sleeping room. Ninety-two per cent of the families had an inadequate milk supply and 48 per cent had less than half the milk that they needed. Physical defects known to the mothers to need immediate attention were reported for one-third of the children, but arrangements for correction had been made for only about half of these.

Studies of Physical Fitness of School Children.—The need for more reliable and simpler methods of evaluating the health of school children led in recent years to the development of a number of new indices of nutritional status (among them the "ACH," the Lucas-Pryor, and the McCloy) to replace the index of weight for height and age. The weight-height-age index as the only measure for judging nutritional status of individual children has long been recognized as of questionable value. It has been useful only as one guide to the physician making a physical examination. The use of the usual routine physical examination by physicians for classifying children according to their nutritional status has also been questioned because of the great variability in standards used by physicians and the lack of objectivity in their observations.

With the purpose of attempting to compare and evaluate the various methods of estimating the physical fitness and nutritional condition of school children now in use by school physicians, nurses, and teachers, or recommended for such use, a study was undertaken in New Haven, Conn., in the fall of 1934 by the Children's Bureau, the Institute of Human Relations of Yale University, and the Department of Pediatrics of Yale University School of Medicine, in cooperation with the Depart-

ments of Education and Health of the city of New Haven. Physical examinations, weights and anthropometric measurements of approximately 1,000 children have been made when the children were six years old and again at seven years. The material is now being prepared for statistical analysis.

Study of Physical Status of Indian Children.—A study of the general physical status of 219 Indian children in certain pueblos in New Mexico and Arizona was made in the summer and fall of 1934, in cooperation with the United States Office of Indian Affairs, the Carnegie Institution of Washington, and the Yale University School of Medicine. The children, 103 boys and 116 girls, were between the ages of seven and eleven years.

In addition to being given a physical examination each child was weighed and the crown-heel length and head circumference were measured. Roentgenograms of the lungs were taken. Mantoux (intradermal tuberculin) tests and Wassermann tests were made, as well as the usual routine blood counts and urine examinations. The results of the study will be published shortly.

Comparability of Maternal Mortality Rates.—A report on a study of the comparability of maternal mortality rates in the United States and certain foreign countries, initiated by a committee of the White House Conference on Child Health and Protection and carried on with the cooperation of sixteen foreign nations, was published by the Bureau in 1935.[10]

The report demonstrated clearly that: "Differences in methods of assignment are insufficient to explain the high maternal mortality rate of the United States as compared with foreign countries. The official figure of the United States which in the last few years has exceeded that of every country (sixteen included) except Scotland, remains high no matter what method of assignment is used. Even if the method of the country assigning the smallest proportion of deaths to the puerperal state were in use in the United States, the United States figure would still exceed that of all the 16 countries [included in the study] except Australia, Canada, Chile, and Scotland."

The report made two recommendations:

1. "It would be desirable for the United States Bureau of the Census to publish annually a table showing deaths associated with pregnancy and childbirth by primary and contributory cause and also a table showing deaths associated with pregnancy and childbirth by states, by color, in urban and in rural districts in the states.

2. "An investigation of the differences in assignment procedure and the formulation of rules uniformly acceptable to important countries should be undertaken by the International Statistical Institute at an early date. International uniformity in assignment procedure is the first goal. This uniformity is an essential basis for reliable statistical judgment as to the comparative effect upon maternal mortality of such factors as geographical conditions, physiological characteristics, socio-economic factors, obstetric practice, and community health provision."

Stillbirth Study.—With the cooperation of the American Public Health Association's Subcommittee on Stillbirths and of individual hospitals, the Children's Bureau has under way a study of stillbirth mor-

[10]Tandy, Elizabeth C.: Children's Bureau Publication, No. 229.

tality in hospitals, the purpose of which is to obtain statistical information regarding fetal and maternal conditions associated with fetal mortality, to make possible the development of a classification of causes of stillbirths (fetal and maternal), and to further the development of a special certificate for the registration of stillbirths which may serve as a basis for comparable statistics for the various states.

A schedule has been prepared which contains, in addition to the information on the standard stillbirth form for reporting now in use in many states, items of additional information of a medical nature. Two physicians are visiting the hospitals with large obstetric services throughout this country to obtain their cooperation in filling in the schedules and returning them to the Bureau for analysis. Records of 5,000 stillbirths will be obtained in this way.

Other Publications.—In addition to these special studies the Children's Bureau issued a number of new publications[11, 12] during the year 1935 and a number of revisions of former publications such as *Prenatal Care, Infant Care,* and *The Child from One to Six; His Care and Training.* It has also made available various folders dealing with maternal and child health and charts portraying various aspects of Infant and Maternal Mortality.

THE SOCIAL SECURITY ACT AND CHILD HEALTH

The following quotation[13] gives briefly the principal provisions of the Social Security Act which apply to child health, as approved by Congress, August 14, 1935:

> The Social Security Act, in title V, parts 1, 2 and 3, authorizes federal grants to the states (including Alaska, Hawaii and the District of Columbia) for maternal and child health services, services for crippled children, and child welfare services. Federal administration of these provisions is placed by the act in the Children's Bureau of the United States Department of Labor. State administration of the maternal and child health provisions is placed under the state health agency, that for services for crippled children under whatever state agency is designated for that purpose by the state itself, and that for child welfare services under the state public welfare agency.
>
> The purpose of the maternal and child health services, as stated by the act, is to enable each state "to extend and improve, as far as practicable under the conditions in such state, services for promoting the health of mothers and children, especially in rural areas and in areas suffering from severe economic distress." A total annual federal appropriation of $3,800,000 is authorized by the act for this purpose. Allotments to the states from this appropriation are to be made as follows: (1) to each state a uniform grant of $20,000 to be matched by the state; (2) to each state such part of $1,800,000 as the number of live births in such state bears to the total number of live births in the United States, such grant also to be matched 50-50 by the state; (3) from the balance of $980,000, funds may be allotted on the basis of the financial need of each state for assistance in

[11]Maternal and Child Welfare Bulletin, No. I, grants to states for maternal and child welfare under the social security act.

[12]Lenroot, Katharine F.: National Aspects of the Social Security Program as They Pertain to the Children's Bureau. (Reprinted from Am. J. Pub. Health, December, 1935.)

[13]From the Report of the meeting of the Advisory Committees on Maternal and Child Welfare Services under the Social Security Act, J. A. M. A. 106: 51, 1936.

carrying out its state plan, taking into consideration also the number of live births; grants allotted from this third fund do not have to be matched by the state.

The services for crippled children are "for the purpose of enabling each state to extend and improve (especially in rural areas and areas suffering from severe economic distress), as far as practicable under the conditions of such state, services for locating crippled children and for providing medical, surgical, corrective and other services and care, and facilities for diagnosis, hospitalization and after-care for children who are crippled or who are suffering from conditions which lead to crippling." A total annual federal appropriation of $2,850,000 is authorized for this purpose, to be allotted to the states as follows: "$20,000 in the form of a uniform grant to each state, and the balance to be divided among the states according to the need of each state, . . . taking into consideration the number of crippled children in such state in need of the services referred to . . . and the cost of furnishing such services to them."

In order to receive these funds, a state must submit to the Children's Bureau for approval a state plan for maternal and child health services and a state plan for crippled children's services. Conditions to be met by state plans are as follows:

1. Financial participation by the state.
2. Administration or supervision of administration by the designated state agency.
3. Such methods of administration (other than those relating to selection, tenure of office and compensation of personnel) as are necessary for efficient operation of plan.
4. Provision for such reports by the state agency as the Secretary of Labor may require.
5. For maternal and child health services: extension and improvement of local maternal and child health services administered by local child health units. For crippled children's services: provision for carrying out the purposes specified in the act.
6. Cooperation with medical, health, nursing and welfare groups and organizations and, in the case of the crippled children's services, "with any agency in such state charged with administering state laws providing for vocational rehabilitation of physically handicapped children."
7. And in addition, for maternal and child health services: provision for development of demonstration services in needy areas and among groups in special need.

Two other phases of the Social Security Act are closely related to the maternal and child health and crippled children's programs, namely, the federal grants for the extension of child welfare services administered by the Children's Bureau and the aid to dependent children in their homes administered by the Social Security Board.

The child welfare services are established by the act "for the purpose of enabling the United States, through the Children's Bureau, to cooperate with state public welfare agencies in establishing, extending and strengthening, especially in predominantly rural areas, public welfare services . . . for the protection and care of homeless, dependent and neglected children, and children in danger of becoming delinquent." The total annual federal appropriation authorized for this purpose is $1,500,000, to be allotted $10,000 to each state and the remainder on the basis of the ratio of the rural

> population of the state to the total rural population of the United States. The act does not require that these grants be matched by the state in any specified ratio. The funds are available for assistance to state agencies and for payment of part of the cost of local service. In the case of the child welfare services the act states that the plans are to be "developed jointly by the state agency and the Children's Bureau" and that the funds "shall be expended for payment of part of the cost of district, county or other local child welfare services in areas predominantly rural, and for developing state services for the encouragement and assistance of adequate methods of community child welfare organization in areas predominantly rural and other areas of special need."[12]

For the purpose of enabling each state to furnish financial assistance to needy dependent children, an appropriation of $24,750,000 is authorized for the fiscal year 1936, and such sums as may be necessary thereafter, to be used for making payments to states which have state plans for aid to dependent children approved by the Social Security Board. The term "dependent child" is defined to mean a child under the age of sixteen years who has been deprived of parental support or care by reason of the death, continued absence from the home, or physical or mental incapacity of a parent, and who is living in the home of his father, mother, or other relative or relatives, as specified in the act.

States with approved plans will be reimbursed to the extent of one-third of the total expenditures, with the exception that the state or local administrative unit will bear the full cost of any payment in excess of $18 per month for any dependent child or, if there is more than one dependent child in the same home, in excess of $18 for one such child and $12 for each other child.

The Secretary of Labor appointed a General Advisory Committee on Maternal and Child Welfare (Kenneth D. Blackfan, M.D., chairman), and special committees on Maternal and Child Health, (Henry F. Helmholz, M.D., chairman), Crippled Children (Albert H. Freiberg, M.D., chairman), and Child Welfare Services (H. Ida Curry, chairman) to assist the Children's Bureau in the administration of the maternal and child health and welfare phases of the Social Security Act. These committees met on December 16 and 17, 1935, and made recommendations concerning the plans to be made by the states for submission to the Children's Bureau. Subsequently a special Advisory Committee on Maternal Welfare was organized with Fred L. Adair, M.D., chairman. The membership of these committees is such as to bring to them the best judgment of the various medical specialties concerned and to give wide national representation. The reader is referred to the report of these Committees[13] for the names of those who participated and for the recommendations adopted. Its careful study is recommended for an understanding of the policies upon which plans are being formulated. The general nature of these policies may, however, be suggested by the following quotations:

> ". . . . there must be a great deal of leeway allowed in the development of these plans but . . . certain features with regard to organization and personnel are essential in any plan."
>
> "It was the consensus that there should be in the state department of health a division of maternal and child health or a comparable administrative unit, coordinate with all other major administrative divisions, with a

director responsible to the health officer. It was further suggested that the director should be a physician and that additional medical staff for consultation and advisory service should consist of full-time or part-time physicians with training and experience in either maternal or child health work, preferably both. It was suggested that a full-time dentist be added to the medical staff and, furthermore, that there might be regional advisers to professional groups in the fields of pediatrics, obstetrics, and dentistry."

"In view of the fact that the number of qualified persons available for positions of this kind is probably not large enough to meet the need, the committee approved the use of funds for the training of personnel.

"As regard participation in a maternal and child health program by local or other qualified physicians, the committee was of the opinion that such services should be arranged for jointly by the local health department and the local medical association, with the advice of the director of the state division of maternal and child health.

"It was the opinion of the committee that as far as possible the maternal and child health work in any given area should be carried on by local qualified physicians and, where such are not available, that other arrangements be made in local maternal and child health centers.

"The committee also agreed that the medical men taking part in this program should be paid for their services."

"The committee emphasized strongly the importance of the educational features of the program and suggested that the divisions of maternal and child health services in the several states might well be coordinating agencies for all health education concerning mother and child.

"The committtee heartily endorsed the publication of a brochure that is in process of preparation by the Children's Bureau relative to its organization, general functions, and aims, with special emphasis on the supervision of its medical activities by physicians, and it further recommended that the medical profession be kept constantly advised of the progress and development of these activities through the medical press."

"The division of the United States into from five to seven regions, each with regional representatives headed by physicians, will provide consultative field services to the states in problems of maternal and child health and crippled children. Each regional office will also have the services of public health nurses and social service consultants for education and consultative work in connection with child health and child welfare. Other state and regional consultants, such as trained orthopedic surgeons or technical advisers, will be used as required."

"The committee discussed the need for state leadership in carrying out the requirements of the act and the necessity for adequate supervision by the official agency when administration is in the hands of local political subdivisions. It was felt that if present administration of a program by a state agency was satisfactory, every effort should be made to preserve or maintain this."

"It was the sense of the committee that it would be desirable for the American boards of certification to make available to the states suggestions as to qualifications for physicians and surgeons to serve in the various states in connection with services for crippled children under the Social Security Act and act in an advisory capacity to the states as requested."

"The committee was of the opinion that physicians performing services under this act should receive remuneration for such services and that policies governing such remuneration should be worked out jointly by the state and local medical societies and the state agency administering the program. The committee recommended that the states set up professional advisory committees representing the medical, nursing, physical therapist and social work interests."

"It was urged that special consideration should be given, from both a research and an administrative point of view, to the large group of children with cerebral palsy, especially with respect to their social and educational care."

The Advisory Committee on Maternal and Child Health took cognizance of the report of the Committee of the State and Territorial Health Officers appointed to consider standards for state divisions of maternal and child health, personnel and programs, and their recommendations were in accord with those adopted by this committee.[14]

STATE PLANS FOR MATERNAL AND CHILD HEALTH SERVICES THUS FAR DEVELOPED UNDER THE ACT

Between February 11, the date when the social security appropriations became available and April 26, 1936, thirty-nine states, Alaska, the District of Columbia, and Hawaii had qualified for the federal grants for maternal and child health services, and all but one of the other states had submitted plans. Before the end of the fiscal year, June 30, it is anticipated that all of the states will have taken advantage of the federal offer and embarked upon an expanded maternal and child health program.

Every state which has had its plan approved now has or will soon have a bureau of maternal and child health as a major division of its state department of public health. In many cases the directors of these bureaus are pediatricians or obstetricians, and all such bureaus have or are planning to have medical directors. The state staff frequently includes other pediatricians, obstetricians, and dentists. Public health nurses for maternal and child health work are either included in these bureaus, or, if there is a separate bureau of public health nursing giving generalized nursing service, the bureau of maternal and child health is likely to have assigned to its staff a nurse specially trained for maternal and child health consultation service to state and local public health nurses.

Local plans will be made and carried out under medical direction and, where possible, under pediatricians and obstetricians. Local practicing physicians will be employed for part-time service.

More than twenty-one states are planning service to physicians through institutes, refresher courses, and other means of keeping them abreast of the latest medical knowledge in obstetrics and pediatrics.

The grants under the Social Security Act cover maternal and child health as distinguished from those under the Maternity and Infancy Act, which covered only maternity and infant hygiene. Accordingly,

[14]Report adopted by the Committee on June 19, 1935. Copies may be had on request from the Bureau of Health and Public Institutions, American Medical Association.

state plans cover maternal, infant, preschool, and school hygiene and may include work with children leaving school for industry. Under the act also special emphasis must be given to rural areas.

All state plans have one or more similar features aimed primarily at the education of doctors, nurses, parents, and children in the care of the health of the mother and child. The Ohio plan, for example, includes an educational program for county public health nurses on maternal and child hygiene, an educational program for mothers' clubs, Parent-Teachers' Associations, and other organizations, preparation and distribution of literature, a demonstration program in setting up classes in prenatal care, infant welfare stations, and preschool conferences, a monthly bulletin on maternal and child health, consultation services to health districts on maternal and child hygiene, investigations on maternal deaths, infant mortality, correction of physical defects, and measurements of nutritional status, and tuberculosis surveys among high school students.

In order to secure a share of the $980,000 fund each state must show special need for assistance in carrying out its plan.

Many of the demonstrations required under the conditions of the federal act provide for general maternal and child welfare services in particular counties with special state assistance. Such demonstration projects are sometimes made training centers for maternal and child health personnel as in Arkansas, Florida, Kentucky, and West Virginia. Such projects are intended to win the counties served and other counties to the support of a well-rounded maternal and child health service.

Several states are using their demonstrations in a special effort to reduce maternal mortality by providing one or more of the following: maternal health education, obstetric service for examination and consultation, and nursing services for prenatal, delivery, and postpartum care. Alabama, California, Connecticut, Florida, and Maine have projects of this type. In New Hampshire, Nashua is to have a nurse to organize a maternal welfare registry and the effort will be made to reach every expectant mother early. The Hospital Obstetric Society of Ohio is cooperating with the State Health Department in a study of maternal deaths in Ohio hospitals.

Other demonstrations include the following: in Arizona a mobile tuberculosis unit to give diagnostic and advisory service to referred children; in Oklahoma a demonstration in five counties with about 25 per cent Indian population giving a generalized public health service with emphasis on maternal and child health under a Children's Bureau pediatrician; and in California nurses are to follow migratory agricultural workers to protect the health of mothers and children.

STATE PLANS FOR SERVICES FOR CRIPPLED CHILDREN

The crippled children's program as it is developed will locate the children who are crippled or who are suffering from conditions which lead to crippling in rural or inaccessible areas and in areas of severe economic distress. It will provide diagnostic clinics by qualified personnel and arrange for medical and surgical treatment in hospitals with increased facilities for convalescent and follow-up care.

In finding ways and means of securing care for all crippled children the provisions of the Social Security Act will supplement the existing services which are rendered by lay and professional groups in public agencies and private organizations.

One year ago many states had no official agency for providing services for crippled children as a recognized public responsibility. In other states having official agencies, the funds appropriated were so small as to be almost negligible. In contrast, this year there has been a tremendous increase in interest in all services of this nature. There are now official agencies in forty-six states and territories, and there is a prospect of several more being established in the near future. Thirty-eight states and one territory have submitted plans to the Children's Bureau for approval. Twenty-nine of these plans have been approved, and several more states will probably have secured approval of their plans before the end of the fiscal year.

The official agency within each state which will administer these services varies. In 16 states with approved plans, the service will be administered by state departments of health; in 10 by state departments of welfare; in 3 by the state departments of education; in 8 by state crippled children's commissions; and in 2 others by state universities or university hospitals.

Notwithstanding the variation in agencies which will administer plans, a common feature of all will be the development of a state-wide program of services for crippled children. In the states with existing programs this will mean the extension and improvement of these services. Most of the states have sufficient reliable case records, gathered in the past through public and private efforts, to initiate extensive plans for clinic and hospital care without further surveys. In some states surveys will be organized to be followed by diagnostic clinics and hospitalization as soon as the children can be admitted. In some states convalescent homes are planned to shorten periods of hospitalization and thus increase the numbers of available beds. In some states an evaluation of the procedure of locating and treating crippled children will be included in the state plan.

As a part of every plan, cooperation with the vocational rehabilitation service of the states is required. Vocational training and placement will be coordinated through joint plans which will allow education and training to proceed with treatment.

A majority of the plans which have been approved provide either for administration by state departments of health or for cooperation with state and local health departments. These services include early reporting of newborn infants with injuries and congenital malformations, the establishment of special laboratory facilities during epidemics, the use of local public health nurses to assist in case finding, diagnostic clinics, and follow-up care, and provision for the training of state and local personnel in the technical problems of care of crippled children through staff education by qualified instructors or by attendance at training centers established throughout the country.

Since the crippled child's need for more than physical restoration must be considered, social workers have been included on the staff in many states to coordinate plans for physical restoration with the service of child welfare agencies. This will make possible the development of social planning for crippled children.

A general advisory committee composed of representatives from the medical, health, welfare, nursing, and educational services is included in a majority of the state plans. Members from private groups and organizations interested in the problems of crippled children have been in-

cluded. A professional group, composed of orthopedic surgeons, pediatricians, hospital heads, and representatives of the state medical association, is usually included as a technical advisory committee. These committees should prove invaluable in giving advice regarding general policies, scope of program, hospital standards and rates, qualifications of personnel, surgeon's fees, arrangements for diagnostic clinics, and provisions for cooperation with the private organizations.

The success of the program in any state will depend on the wisdom shown in developing a well-balanced plan and the cooperation of the interested groups.

COMMENT

The difficulty of finding a sufficient number of well-qualified persons to fill all the newly created positions made available by this act is recognized. Thorough training in pediatrics, obstetrics, or orthopedics is required for most of them in addition to a knowledge of public health problems and procedures. It is evident, however, that an increasing number of recent medical graduates are looking to the public health field as an opportunity for public service and are seeking postgraduate training in order to qualify adequately. Both the Children's Bureau and the United States Public Health Service are apparently making every effort to maintain high standards in the selection of personnel to fill the newly created positions throughout the various states. Owing to the limitation of time and the lack of adequate numbers of fully qualified men and women, the principle is being followed of selecting persons with adequate medical background, even though they lack specific public health training, and of providing for these new recruits special postgraduate training in public health after enrollment in the service. This properly places first emphasis on the need for adequate medical training. It does seem unfortunate to the reviewer, however, that this whole program could not have been developed more gradually and time allowed for the thorough preparation of personnel through adequate postgraduate training.

American Academy of Pediatrics

Proceedings

SIXTH ANNUAL MEETING OF THE AMERICAN ACADEMY OF PEDIATRICS

KANSAS CITY, MO., MAY 11 AND 12, 1936

The sixth annual meeting of the Academy of Pediatrics was held at the President Hotel in Kansas City, May 11 and 12, 1936. Dr. Henry Dietrich, president, presided; 253 members and 87 visitors were registered.

Monday morning, May 11, was given over to Round Table Discussions, which were repeated Tuesday afternoon, May 12.

Subject	Teacher	Assistants
Appendicitis	A. H. Montgomery	Joseph Brennemann
Asthma	Francis Rackemann	Bret Ratner
		Marion B. Sulzberger
Childhood Psychiatry	F. G. Ebaugh	Leo Kanner
		James S. Plant
Parasitic Infections	E. C. Faust	Horton Casparis
		Hugh L. Dwyer
Passive Prophylaxis Against Infection	John A. Toomey	Archibald Hoyne
		Sidney D. Kramer
		E. E. Martmer
		E. B. Shaw
Appraisal of the Child	Harold C. Stuart	
Whooping Cough	E. J. Huenekens	W. L. Bradford
		J. M. Frawley
		Louis W. Sauer
Recent Advances in Nutrition	W. McKim Marriott	S. W. Clausen
		Philip C. Jeans
Roentgenology of the Chest	L. R. Sante	
Sinusitis	L. W. Dean	E. C. Mitchell
The Overweight Child	L. H. Newburgh	Julius Hess
		S. Z. Levine

On Monday afternoon two Panel Discussions were held: one, "Resistance to Tuberculosis in Children" with Chester A. Stewart, chairman, assisted by Louis Dienes, W. H. Frost, J. A. Myers, and H. A. McCordock; the second, "Cyanosis in the Newborn" with A. Graeme Mitchell as chairman, assisted by F. L. Adair, B. E. Bonar, Glenn E. Cullen, and N. J. Eastman.

Monday evening was given over to the annual meeting of the state chairmen.

At the general meeting on Tuesday morning the annual presidential address was given by Dr. Dietrich. This appears on page 657 of this JOURNAL. Following the

presidential address two scientific papers were presented which had been selected by the editors of the *American Journal of Diseases of Children* and the *Journal of Pediatrics* as outstanding among the manuscripts submitted for publication during the past year. The first paper, "The Gastrointestinal Portal of Entry in Poliomyelitis," by Dr. John A. Toomey appears on page 664 of this JOURNAL, and the paper by Dr. R. Cannon Ely, Dr. Arda Alden Green, and Dr. Charles F. McKhann was published in February and will be found on page 135 of this volume.

The various committee reports, including that of the Executive Committee, were presented in mimeographed form, and they follow. The report of the Executive Committee was approved. With the installation of the new president, Dr. Laurence De Buys, the session ended.

Meeting of the Executive Board
Sunday, May 10, 1936

The meeting of the Executive Board was called to order at 10 A.M. in the Hotel President by the president, Dr. Henry Dietrich. All members were present with the exception of Dr. Louis Schroeder who, because of illness, was unable to attend.

The report of audit was approved.

The secretary was instructed to notify delinquent members that Article XII of the Constitution and By-Laws relating to delinquency in the payment of dues would be upheld.

The Executive Board recommends that it be the understanding of the American Academy of Pediatrics that the word "crippled" include any child suffering from physical handicap.

The Executive Board recommends that inasmuch as the newborn infant is a special pediatric problem, it should be entrusted only to those who have special training in this type of work. This applies particularly to teaching centers.

The Board approved the following recommendations of the Committee on Medical Education:

I. Internes. First year of hospital training after graduation from medical school. While usually applied to a rotating service in a general hospital, it also applies to a straight service on a surgical, medical, or pediatric service. Such service does not count as part of a specialized training required for certification in any specialty. Such services are designated under the heading "D" in the report (*Journal of Pediatrics* 7: 427, 1935).

II. Residents (Specialized Training).

a. "Junior" applies to first year of training in the specialty after the interne year or years.

b. "Associate" applies to the second year of specialized hospital training after completing a junior residency.

c. "Chief" applies to a third year of specialized hospital training after completing an associate residency.

These terms should be used officially in designating positions. It might be that the only, or senior, resident on the hospital staff would be a "junior" or an "associate resident." The terms apply to the period of training in the specialty rather than to the executive status of the resident in the specific hospital.

The Board approved the recommendation of the Committee on Medical Education that a study of residencies be undertaken by one or more of the pediatric societies.

The Board accepted with regret resignations of chairmen of the following committees:

William Palmer Lucas, chairman of the Committee on Nursing Education (to remain a member of the committee)

LeRoy Wilkes, chairman of the Committee on School Health and School Health Education (to remain a member of the committee)

Martha Eliot, chairman of the Committee on Child Health Relations (to remain a member of the committee).

The President was authorized to appoint a chairman of the Committee on Nursing Education.

Harold H. Mitchell, Freeport, N. Y., was appointed chairman of the Committee on School Health and School Health Education.

M. Hines Roberts, Atlanta, Ga., was appointed chairman of the Committee on Child Health Relations.

The resignation of Samuel McClintock Hamill, Philadelphia, as representative to the Children's Bureau was accepted with regret, and J. H. Mason Knox, Baltimore, appointed as the representative.

The Board recommends that Dr. Bernard Fantus, of Chicago, be made an Associate Fellow of the Society.

The invitation of the United States Constitution Sesquicentennial Commission to join the celebration of the one hundred and fiftieth anniversary of the formation of the Constitution of the United States was accepted.

The Board recommends that there be created a Committee on Immunization Procedures consisting of four members.

The Board recommends that the following Fellows be added to the Committee on Cooperation with Nonmedical Groups and Societies: Albert D. Kaiser, Rochester, N. Y.; James W. Bruce, Louisville, Ky.; Lee Forrest Hill, Des Moines, Iowa.

It was voted that the next annual meeting of the American Academy be held in Chicago, the time to be decided upon by the Executive Board.

The following action was taken relative to the annual meeting in 1937: That it cover a period of two and one-half or three days instead of two days as heretofore and that commercial and scientific exhibits be held in connection with the meeting.

The following applications were approved:

Region I

Harry Edwin Blair, Hagerstown, Md.
Charles Bradley, East Providence, R. I.
M. Bernard Brahdy, Mount Vernon, N. Y.
Allan M. Butler, Boston, Mass.
Ernest Caulfield, Hartford, Conn.
Sidney Shaw Shipman, Norwalk, Conn.
Samuel W. Clausen, Rochester, N. Y.
Edward P. Essertier, Hackensack, N. J.
Henry E. Gallup, Brookline, Mass.
Louis E. Goldberg, Newark, N. J.
Manuel S. Hirshberg, Hartford, Conn.
Herbert F. Jackson, New York, N. Y.
George Kahn, Boston, Mass.
James DeWitt Kerr, Lebanon, Pa.
Sidney David Kramer, Brooklyn, N. Y.
Sylvan D. Lazarus, Brooklyn, N. Y.
Walter Levy, New York, N. Y.
Herman L. Maslow, Brooklyn, N. Y.
Dorothea May Moore, Cambridge, Mass.
Norman A. Pokorny, Springfield, Mass.
Max Palinsky, Brooklyn, N. Y.
Joseph C. Regan, Brooklyn, N. Y.
Benjamin B. Rittenberg, Philadelphia, Pa.
Carl L. Ruder, Mt. Lebanon, Pittsburgh, Pa.
Harry M. Shapiro, The Bronx, New York, N. Y.
John Howell West, Easton, Pa.
Samuel Wetchler, New York, N. Y.
Raeburn J. Wharton, Johnson City, N. Y.
Louis I. Skirball, Boston, Mass.

Region II

H. D. Long, Chattanooga, Tenn.
W. E. Van Order, Chattanooga, Tenn.
Mercer Blanchard, Columbus, Ga.
Wallace A. Clyde, Fairfield, Ala.
H. F. Garrison, Jackson, Miss.
Edwin A. Harper, Lynchburg, Va.
Marvin C. Overton, Lubbock, Texas

Region III

Joseph A. Bauer, St. Louis, Mo.
Iris McKy Chamberlain, Chicago, Ill.
Carl L. Cohen, Chicago, Ill.
Ethel M. Davis, Chicago, Ill.
Raymond F. Grissom, Oak Park, Ill.
*Forrest A. Harrison, Springfield, Mo.
Joyce I. Hartman, Cleveland, Ohio
Peirce D. Knott, Sioux City, Iowa
Thos. F. Krauss, Rockford, Ill.
J. N. Lande, Sioux City, Iowa
Abe Matheson, Chicago, Ill.
John M. Mitchell, Evanston, Ill.
George F. Patterson, Cincinnati, Ohio
C. H. Schroder, Duluth, Minn.
Frank Seinsheimer, Cincinnati, Ohio
Philip H. Woutat, Grand Forks, N. D.
Paul Edw. Belknap, Topeka, Kan.

Region IV

John Reford Davies, Vancouver, B. C., Canada
Frank H. Gaugin, Honolulu, Hawaii
Robert John Groom, Honolulu, Hawaii
Victor C. McPhee, San Francisco, Calif.
William Anthony Reilly, San Francisco, Calif.

*Died May, 1936.

Report of the Executive Board

As it is customary for the Executive Board to present a report to the general body of the Academy and the responsibility of this report is placed with your incoming president, it devolves upon me, therefore, to submit this report. At the two meetings held by the Board during the past year certain items have occurred to me as being of sufficient importance to be emphasized here.

At the first meeting which was held on Nov. 30, 1935, at the Georgian Hotel, Evanston, Ill., all the members of the Executive Board were present and our president, Dr. Dietrich, presided. There were two actions which were taken and which were significant: First, raising of the dues to make it possible to balance the budget for the year 1936-1937; and, second, the resolutions which were passed following the discussion with the two representatives of the Committee on Allocation of Research Funds, Dr. Thomas B. Cooley and Dr. Fritz B. Talbot. These resolutions have already appeared in the official organ of the Academy, the *Journal of Pediatrics*, but are worthy of reiteration:

1. That the Committee on Allocation of Research Funds shall be reconstituted as the Council for Pediatric Research of the American Academy of Pediatrics.
2. That it would welcome the endorsement of this Council by the American Pediatric Society.
3. That this Council shall consist of five or more members.
4. That one member and not more than one each year shall retire.
   a. That retiring age of each member shall be not greater than seventy years.
   b. Any retiring member is eligible for reelection.
5. That any retiring member shall be replaced by a member elected by the Executive Board of the American Academy of Pediatrics from two or more nominations of the Council.
6. That it is the sense of the Executive Board that as soon as possible the Council shall be organized as an autonomous unit within the Academy.

Eighty-nine additional members were elected to the Academy at the November meeting; this is very gratifying.

Laurence R. DeBuys
Vice President (President-Elect)

Report of the Secretary

Our membership now is fast approaching the thousand mark, and with the Fellows added at this meeting it will undoubtedly top it. We are getting very close to the saturation point. Since the younger men will come in and unfortunately some of our men will pass on, it is not likely that our membership will increase as it has in the past.

The first four or five years of the Academy's existence were taken up with organization and the development of membership. That time is largely past. Gradually and with increasing force the Academy is making itself felt in medicine in this country. One after another the new activities develop as we branch into new fields. The cry is always for more men to meet these conditions, and those men are developing slowly but surely. We pick up a man here and a man there who has shown his ability and his interest. Gradually the burden of the work will fall upon the shoulders of these men who will in turn have to develop their own successors.

During the past year certain significant things have happened. One of the most significant is the formation of the Council on Pediatric Research. It is too early yet to predict what may come out of this effort, but its possibilities are almost unlimited. Wisely, I think, this council has decided to watch its step and go slowly.

Another most significant situation is the approach of the Academy to the Parent-Teachers' Association, an association of a great deal of power and large membership whose influence is very great and which has shown a marked tendency to develop its medical efforts along the proper lines.

These are but a few of the things that have been done. Within the Society the rapid development of state organizations is most gratifying to us all. It is through state organization that we can expect to distribute our ideas and our knowledge. Another effort which has finally crystallized and has shown excellent results is the development of the regional meetings. The secretary has been happy to be able to attend all the regional meetings last fall, and in his opinion each was a great credit to pediatrics in this country. There is interest in these meetings—an interest which is well deserved.

It is my sad duty to bring before this meeting the names of those who have died. As many of you know, Dr. Orville E. Baldwin, of Columbus, Ohio, had an attack of coronary thrombosis and died in the hotel at New York City during the last meeting. The other deaths which we have suffered are thouse of Dr. Morgan Smith, of Little Rock, Ark., who was state chairman for that state and organized pediatrics there; Dr. Carl K. Wagener, of Pittsburgh, and Dr. William B. McClure, of Evanston, Ill.

Clifford G. Grulee
Secretary

Report of Region I

It is becoming increasingly evident that Academy members can accomplish a great deal if they will actively ally themselves with their state societies. In the northeastern states; varying in size as they do from Rhode Island to New York, no set approach to the problems of infant and child hygiene is possible. Irrespective, however, of the size of a state, every Academy member must realize that the attention paid to infant and child welfare by his state society with very few exceptions will be in proportion to the pressure exerted by its membership.

Perhaps the best example of this in the East is to be found in New Jersey. Under the leadership of Stanley Nichols, there has been worked out a cooperative working relationship between the State Department of Health and the medical profession of New Jersey, which, while it may cause doubts in the minds of those who are completely sold on the idea of full-time personnel for all public health work, is a sane,

safe program. Both the maternal and child health set-ups are concerned not only with the problems of the mother and child, but with the education of the physicians, going so far in the latter instance as to set aside a special fund for the education of the physicians in rural areas where none are qualified to carry on the local maternal and child health activities. Besides this special interest, the New Jersey men held a successful clinical meeting in Newark last fall and will hold a special meeting in June at the annual meeting of the state society. There has also been a steady drive for new members and a continued interest in the development of plans for the postgraduate instruction of the general practitioner in pediatrics.

In New York state there is a special Domestic Relations Act, which sets aside stated sums for physically handicapped children. This is administered under the direction of the State Departments of Health and of Education. Thus far the only recipients of aid have been orthopedic cripples. There is set aside in the Social Security Act a great deal of money for what is described as "crippled children." This failure to define more clearly what a crippled child is, is understandable since it represents the thoughts of legislators. It seems wise to have the Academy of Pediatrics lend the weight of its authority to a broader conception of the term "cripple," and the New York state members make this recommendation to the Executive Committee.

A complaint received from one New York member stated that members of the home economics department of a certain university were sent out to tell mothers how to feed babies. These are not nurses and the question was raised how widespread this particular practice is and whether or not it should meet with approval. The particular complaint is being investigated, but no report is yet available.

In Connecticut special committees have been appointed to deal with postgraduate instruction, public institutions which deal with children, legislative and coordination to work with the State Department of Health on matters outside the social securities program.

In Maryland the Social Security Act is operative, but to date it awaits approval in New Hampshire and Vermont.

The increase in the membership of Region I continues to be satisfactory, and there can be but little doubt that the influence of the Academy continues to grow in every way.

Louis C. Schroeder
Regional Chairman

Report of Region II

Every state during the past year has initiated some new form of pediatrics activity. It has been found advisable by most of the state chairmen to work in conjunction with the already established societies and other agencies. As a result, much better cooperation is received, and less friction is created. The fact has become known that the function of the Academy is to promote child health in its broadest sense, and the Academy as a unit and its members individually are always available when called upon.

An enumeration of some of the activities would include the following: First, a committee was appointed with Dr. W. C. Davison as chairman, to investigate children's hospitals in this region, including both the care of the patient and the hospital records. It was also the function of this committee to determine why more children's hospitals were not recognized as teaching institutions by the Committee on Hospitals of the American Medical Association. This committee has been quite active and is now assembling data which will be reported in the near future. As a result of this activity, several new hospitals in the region have been accredited.

Second, postgraduate instruction in pediatrics is being promoted in most states in conjunction with the state medical societies. Kentucky, particularly, has been active and offers a postgraduate course to be given in Louisville, beginning Wednesday, April 22, 1936, and continuing each Wednesday for ten weeks, from 9 A.M. to 1 P.M. One hour will be spent each week in a discussion by the staff of all interesting cases in the hospital. All of the newer methods of treatment by transfusion, lumbar and cisternal puncture, hypodermoclysis, peritoneal injection, etc., will be demonstrated on patients. Lectures will be given on many puzzling problems in diagnosis and treatment, and such other questions as may be suggested will also be discussed. The cost will be only $5. I am reporting this method in full because it appears to be such an excellent approach to postgraduate training in pediatrics for rural physicians.

Tennessee, through the State Medical Society, has a Committee on Postgraduate Education; this is subdivided into the various specialties. The one on child health has a pediatrician appointed in each third of the state to supervise his particular locality. Under a general plan selected pediatricians are sent to the various local groups. A number of talks throughout the state have recently been made on the subjects, "Immunity" and "Care of the Well Child." This plan has proved satisfactory up to the present time.

Details of specific activities recently initiated have not been received from the other states.

All of the states in this region have at the present time a state pediatric society. In some instances this is a part of the state society and in others, a separate group. By working with the state society and the local societies throughout the state, this group is able to have one meeting a year at which various pediatric topics of interest to the general practitioner are discussed, and, in addition, throughout the year they send pediatricians to appear on the programs of the local groups. This method has, undoubtedly, created interest in pediatrics. Wherever possible, particular stress has been laid on prenatal care and care and immunization of the well child. As a result of these activities, every state is showing a much larger proportion of immunized children each year.

Regional Meetings.—The Southern Medical Association meets in Baltimore, Md., November 17 to 20. Region II has been invited to meet with Region I in Baltimore, Monday and Tuesday, November 16 and 17. Dr. Schroeder, chairman of Region I, states that the program will be conducted under the auspices of the pediatricians of Baltimore. This will be our second joint regional meeting. The one held in St. Louis last year was most successful. It was the first time in the history of the organization that all of the state chairmen were present. There were 67 members from Region II, exclusive of St. Louis, registered. These joint meetings give us an opportunity to renew many old acquaintances and form new ones. I am sure we are deeply indebted to Region I for the invitation and hope we have a large attendance.

Dr. Loranz, secretary of the Southern Medical Association, informs me that hotel accommodations are limited in Baltimore. For this reason, it would be well to make reservations early.

There has been considerable controversy as to the best time to hold the regional meeting each year. The request was made by Dr. Loranz and Dr. M. Hines Roberts, secretary of the Pediatric Section of the Southern Medical Association, to consider the advisability of having our regional meeting on the same day as the section. If the section meets in the morning, the regional meeting can be held in the afternoon, the benefit being that it would probably increase the attendance of both meetings as many of the men would make an effort to attend both if they did not have to remain away from home so long.

We are deeply indebted to Dr. Loranz and the officers of the Southern Medical Association for their many favors in the past. They have always given us publicity

in the *Southern Medical Journal* and have shown us every courtesy. I, personally, feel that the Academy should comply with their wishes as far as we possibly can.

This contemplated change is published in this report because I believe it should be discussed by all the members rather than be determined by the officers and the state chairmen of the Academy.

Suggestion for the conduct of the regional business meeting: Last year in St. Louis many of the Fellows attending the session did not come to the regional business meeting. It is felt by the officers of the Academy that this is a most important phase of our regional activity. Perhaps the reason the men do not attend is that the meetings in the past have not been conducted in a way to make them attractive or in a way to make the members feel that they are a part of the Academy and should have some voice in governing its policies. For this reason, it has been suggested by your president-elect, Dr. DeBuys, that next year the reports of the state chairmen be sent to the regional chairman not later than October 1. That these reports be condensed and mimeographed, and sent to the members before the regional meeting. This would give each member some idea as to what is taking place in the various states, and give the time that is now used in reading the various reports for discussion. This is offered as a suggestion.

Changes in Personnel.—There has been only one change in state chairmen this past year. Dr. A. C. Kirby has been appointed in Arkansas to succeed Dr. Morgan Smith, who died.

No other deaths in the personnel of the Academy have been reported to this office.

In closing, the regional chairman would like again to call to the attention of the state chairmen and the members that applications for membership must be in his office at least two months prior to the meeting of the Executive Board in order to give them consideration; and further, that each application must be endorsed by two members of the Academy, and each of the endorsers must send a letter of recommendation direct to the regional chairman. This will save lots of confusion and embarrassment.

I would like to thank the regional board, state chairmen, and members for their support during my term of office, and to assure you that it has been a pleasure to serve.

Edward C. Mitchell
Regional Chairman

Report of Region III

The membership of Region III as of Jan. 1, 1936, was 310. This represents an increase of 82 over the same date of 1934. At the present time 19 applications have been approved by the regional committee, and the individuals recommended to the Executive Board for membership. At the present time we have no Canadian members of the Academy from the two Canadian provinces of Manitoba and Saskatchewan. It is hoped that during the next year a number will be added to this list. It seems likely that from now on the increase in the membership of Region III will be rather small, although there are still some states in which a number of men whom we would welcome in the Academy have not joined.

Dr. Edgar E. Martmer was appointed state chairman for Michigan during the past year in place of Dr. Bernard Carey, who resigned. Dr. Ralph E. Pray was appointed chairman for North Dakota and, until Dr. Ruth Mahon was elected in the fall, was the only member from North Dakota.

The meeting of the Southern Medical Association in St. Louis made it possible to hold a joint meeting of Regions II and III. An excellent program was prepared under the chairmanship of Dr. T. C. Hempelmann with the assistance of the local group. All meetings were joint except the business meetings of the two regions. The

minutes of these meetings as well as the reports of the state chairmen have been published in the February issue of the *Journal of Pediatrics*. At this point I want only to state the great interest displayed by the membership in the work of the Academy and the working out of its various problems. The question of the mode of appointment of state chairman was brought up, and a recommendation was made that he be selected by the state group. The questions of the admission of pediatricians to membership who were not devoting their entire time to pediatrics was discussed. The interpretation of the rules on admissions in Region III has been more lenient than in Region II as applied to small communities. Men who are not devoting themselves entirely to pediatrics have been admitted at the request of the local state members of the Academy. The other thing of special interest was the presentation of the report of the Committee on State Programs presented by Dr. Maurice Blatt, state chairman of Illinois.

The state chairmen of the two regions met together at a Monday luncheon meeting and had opportunity to discuss a number of their problems.

The annual clinical meeting for 1936 will be held in Cincinnati under the chairmanship of Dr. A. Graeme Mitchell on Friday and Saturday, November 20 and 21.

The most important problem before our different states at the present time is the working out and the proper functioning of a program under the Social Security Act. As was pointed out at a recent meeting of the Minnesota State Advisory Committee by Dr. Chesley, our state health officer, the expansion of work for mother and child in forty-eight states will throw a tremendous load on new and oftentimes inexperienced personnel. Our state organizations should be in a position to give very valuable aid in many aspects of the work. In the last week in March funds appropriated for work in the State Bureau of Maternal and Child Health have been received by the State Department of Health for work in states that have had an approved plan of work. It behooves state chairmen to become as conversant as possible with all the work planned and to judge its effectiveness. It should become a major item of business at the state chairmen's meeting in the fall.

Henry F. Helmholz
Regional Chairman

Report of Region IV

In the far West there has been a steady increase in membership in the Academy and constantly increasing interest in its purposes. The annual meeting of the region was held in Seattle, August 9 and 10, Dr. Jay I. Durand being chairman of the scientific program. There was a large and enthusiastic attendance of the membership, and we were fortunate in having as guests Dr. Clifford Grulee, Dr. Borden Veeder, and Dr. Franklin P. Gengenbach. An extremely interesting scientific program was presented by the local membership; this program was previously reported in the *Journal of Pediatrics*. From the standpoint of attendance, interest and enjoyment, this meeting established a new high mark. The next annual meeting will be held in San Francisco, October 22, 23, and 24, 1936, the program being supplied by the San Francisco membership.

Several new appointments of state chairmen have been made during the past year. These include Dr. Harold C. Joesting, Montana; Dr. William A. Shaw, Nevada; Dr. James W. Rosenfeld, Oregon; Dr. Eugene H. Smith, Utah; Dr. Howard Spohn, British Columbia; and Dr. Douglas B. Leitch, Alberta. There have been a number of new members including three from Canada, Dr. Harold W. Price, Calgary, Alberta; Dr. Douglas B. Leitch, Edmonton, Alberta; Dr. Alfred H. Spohn, Vancouver, British Columbia. A number of new applications will be presented at the annual meeting.

From recent reports of state chairmen, it is evident that in all of the various states strenuous efforts are being made by Academy members to promote numerous activities for child welfare and especially to attempt to direct the efforts recently initiated under the Social Security Act in this respect. This latter is being undertaken with some slight confusion, but in Oregon, Washington, Utah, and California professional advisory committees under this Act have been set up, which include in their membership the state chairmen and other members of the Academy.

In this region, in which distances between pediatric centers are so great, most of the problems of Academy activity are being undertaken separately by the various states. This is rendered especially difficult in those states in which there are very few pediatricians and the Academy is represented by only one or two members. Even in these most isolated regions, however, worthwhile activity is being diligently carried out. Dr. Joseph Palma has recently forwarded two applications for membership from Honolulu, and reports that there monthly pediatric meetings are being held for the discussion of community problems. Dr. Harold Joesting, the only member in Montana, reports the establishment of speech correction clinics for school children, establishment of a study group for the exceptional child, and efforts which are being made to establish the first pediatric ward in Butte. Dr. William A. Shaw, the only member in Nevada, is carrying forward work in educational and preventive pediatrics and attempting to initiate activity under the Social Security Act. Dr. Meldrum K. Wylder reports that a division of Child Health has been added to the Bureau of Public Health under the Social Security Act and by means of eight additional public health nurses and with the cooperation of the United States Service an especial study of infantile summer diarrhea, which was inaugurated last year, is being carried forward on a more extensive scale. In Utah the state chairman, Dr. Eugene H. Smith, reports that activity under the Social Security Act has been initiated with the development of a nursing service; a member of the Academy serves on the State Board of Health. An informal study group has been organized by members of the Academy, including in its meetings all those interested in pediatrics; this group has devoted itself to clinical study and to a study of handicapped children and the state institutions caring for them.

In many localities local groups, in which Academy members are at least the nucleus, have organized for the purpose of considering both scientific and community problems. This is true in Salt Lake City, Seattle, Wash., San Francisco, Los Angeles, and San Diego. The San Francisco Association of Members of the Academy of Pediatrics has numerous meetings to which others interested have from time to time been invited, although membership in this association is limited to members of the Academy. One meeting of this group was addressed by Dr. Edith Sappington regarding the purposes of the Social Security Act and their relation to the Academy. At a recent meeting the membership and many guests were addressed by Dr. Charles R. McKhann on his return from the Orient regarding the preparation and use of placental extract.

I wish to express my appreciation of the interest and cooperation displayed by the state chairmen and by the regional committee in the problems of the Academy in this region.

Edward B. Shaw
Regional Chairman

Report of Committee on Child Health Relations to the Executive Board

The Committee on Child Health Relations met for an all day session in Kansas City, May 10, 1936, to consider problems in the field of child health and welfare, especially those arising in connection with the administration of the Social Security Act.

As the first order of business, the committee received from Dr. McCown of the United States Children's Bureau a report of the activities of the federal and state governments under Title V of the Social Security Act, which are now in operation and have to do with maternal and child health services and with services for crippled children.

The report may be briefly summarized as follows:

The Social Security Act became effective on February 11, 1936, when the Supplemental Appropriation Act was signed and funds for grants to states became available.

In conformity with Title V, Part One, of the Social Security Act (grants to states for maternal and child health services), forty-seven states, the Territory of Alaska, the District of Columbia, and the Territory of Hawaii have submitted state plans for maternal and child health services. Of the 50 plans submitted, 42 have been approved and are in operation. Only one state has not submitted a plan, and that because no state funds were available for matching purposes. It is anticipated that by the end of the fiscal year, June 30, 1936, all forty-eight states, the District of Columbia, and the territories will have plans approved and in operation.

We feel that definite public health progress has been made possible in the states through grants-in-aid in adding qualified personnel to the state staff. In June, 1934, there were 22 states with physicians as directors of maternal and child health. Today there are 37. In 26 there were no directors and in 17 there were no divisions of maternal and child health in the state health department organization. Today in the 42 states with plans approved all have or soon will have bureaus or divisions of maternal and child health as major divisions of the state health departments under physicians as directors. In many cases, and this is especially true of states adding new personnel, the directors are pediatricians or obstetricians.

In fifteen states recently appointing directors of maternal and child health divisions, 11 of the appointees are pediatricians. The staff frequently includes other pediatricians, obstetricians, and dentists.

Local plans for maternal and child health services should be made and carried out under medical direction and, where possible, under pediatricians and obstetricians. Local practitioners may be employed for part-time service. In many states, the outstanding pediatricians and obstetricians have consented to act in a part-time capacity in assisting the state in the formulation and operation of its plan.

In respect to the educational program, the Children's Bureau encourages the use of funds for this purpose from grants-in-aid. In more than half of the plans approved, the states are contemplating educational services to physicians through institutes, refresher courses, and other means of keeping the physicians in touch with the latest medical developments in obstetrics and pediatrics.

The state plans for maternal and child health services and education vary widely in the several states.

We welcome this variation, being sensible to the fact that as health needs and health facilities differ in the several states so must the state plans differ to meet these needs.

Summary.—The plan for maternal and child health services is a state plan. The federal government does not propose to substitute something new for something old. The aim of the grants is to supplement; the purpose is to enable the state to extend and improve its existing services to mothers and children.

Funds from federal grants are used to pay part of the cost of the program. The Children's Bureau has responsibility for approving the plan as submitted.

Otherwise, its rôle is consultative. In addition, the Children's Bureau will conduct certain special demonstrations and research and will furnish reports so that the results of maternal and child health activity in the several states may be made available.

The program, then, is one of health education, health services, and health investigations, depending for its success on understanding and cooperation among all groups interested in the health and welfare of mothers and children.

Following this report the committee discussed ways and means by which the state Academy groups might assist most effectively in the development of sound programs and presents the following recommendations to the Executive Board of the Academy:

1. The Committee recommends
   (a) That each state chairman appoint a member or members of the Academy to cooperate with the various state agencies administering the services for maternal and child health, for crippled children and for child welfare in the state;
   (b) That these appointees be the representatives of the Child Health Relations Committee in each state; and
   (c) That the appointments be approved by the Committee on Child Health Relations and by the Executive Board.
2. The Committee recommends that the Children's Bureau be requested to prepare for the Committee a statement of the activities of the Bureau and of the states under Title V of the Social Security Act and that this statement be circulated to all state chairmen with suggestions from the Committee as to possible activities of the Committee representatives in cooperating with the various state agencies administering the program.
3. The Committee further requests the Executive Board to authorize a midwinter meeting of the Committee, since it is not possible to carry out the work of the Committee with but one meeting a year.

Report of the Committee on Cooperation With Nonmedical Groups and Societies

The committee has centered its attention on working out a cooperative plan with the National Congress of Parents and Teachers. During the year a number of meetings have been held with the president and members of its committee regarding a closer cooperation between the two organizations. For some time Dr. Margaret Handy has been an active member of their advisory committee on "summer roundup." The organization of the Academy by states was explained and the possibility of cooperation in plans for "summer roundup" as well as a continuation of the program emphasized. More recently we have been working with them on a series of twenty-six radio talks on growth and development.

Cooperation in an advisory capacity regarding articles of a medical nature in their magazines has also been worked out.

The Committee feels that there is no greater field at present available to create better feeling among the laity toward the medical profession than by cooperative effort in the health work of the various agencies interested in the child.

H. F. Helmholz, Chairman
C. Anderson Aldrich
Margaret I. Handy

Report of the Committee on Hospitals and Dispensaries

During the past year, 1935-1936, the Committee on Hospitals and Dispensaries has cooperated with the Committee on Medical Education to prepare and publish a report giving detailed information concerning house officer positions including interneships, assistant residencies, and residencies in pediatrics among credited children's and general hospitals in this country and Canada.

The Committee at the present time is preparing a report which will present detailed information concerning contagious disease hospitals of the United States and Canada. The first section of this report will appear in early issues of the *Journal of Pediatrics.*

George F. Munns, Secretary
Clifford G. Grulee, Chairman
Murray H. Bass *Henry Dietrich*
Laurence R. DeBuys *Lewis Webb Hill*

Report of the Committee on Infantile Paralysis

The Committee on Infantile Paralysis through its chairman has kept in close touch with the work of the Georgia Warm Springs Foundation, acting in the capacity of consultant to the Board.

It may be stated that the Trustees of the Georgia Warm Springs Foundation are anxious to continue all its work in the most proper and ethical manner. The resources of the foundation are intended primarily for the use of physicians and particularly of orthopedic surgeons, a committee of whom acts in an advisory capacity.

There have been no other activities of this committee since the last meeting of the Academy.

A. Graeme Mitchell, Chairman
D. Lesesne Smith
John A. Toomey

Report of the Committee on Legislation

The Committee on Legislation of the American Academy of Pediatrics, appointed by President Dietrich in September, 1935, has considered only one matter of legislation up to the present time, namely, Public Law No. 271.

Official copies of this law, which is known as the Social Security Act, adopted by Congress and approved by the President August 14, 1935, were furnished members of the Committee on Legislation, together with the official Bulletin No. 1 on Maternal and Child Welfare published by the Children's Bureau, Department of Labor, in explanation of the provisions of the law.

Further extensive explanations and criticisms of the Act were published at various times in the *Journal of the American Medical Association* and a detailed exposition of its provisions set forth in a publication from the Bureau of Legal Medicine and Legislation in the March number of the *Bulletin* of the American Medical Association.

The attention of members of the Academy is called to these various expositions of the Social Security Act and its bearing upon members of the medical profession.

It was the sense of the Committee on Legislation that the degree of participation of the members of the Academy in the administration of the provisions of

this law would fall within the province of the various state chairmen, and other consultants who might from time to time be called upon by the Department of Labor to aid in carrying out the provisions of this new legislation.

There are at present no matters of new legislation now pending before the Committee.

Joseph S. Wall, Chairman
Isaac A. Abt *Philip Van Ingen*
Jay I. Durand *Alfred A. Walker*

Report of the Committee on Medical Education

In conjunction with the Committee on Hospitals and Dispensaries a detailed list of pediatric house officer positions was prepared and published in the September number of the *Journal of Pediatrics* (7: 427, 1935).

In collecting this information considerable confusion was found in the titles given to the house officer positions by the hospitals. As residents are constantly changing from one hospital to another and as it has become necessary for purposes of certification to determine the exact amount as well as the quality of training, the committee recommends that the nomenclature of the Council on Medical Education of the American Medical Association be adopted by all hospitals having residencies in pediatrics.

I. Internes. First year of hospital training after graduation from medical school. While usually applied to a rotating service in a general hospital, it also applies to a straight service on a surgical, medical or pediatric service. Such service does not count as part of the specialized training required for certification in any specialty. Such services are designated under the heading "D" in the report mentioned above.

II. Residents (Specialized Training).

1. "Junior" applies to first year of training in the specialty after the interne year.
2. "Associate" applies to the second year of specialized hospital training after completing a junior residency.
3. "Chief" applies to a third year of specialized hospital training after completing an associate residency.

These terms should be used officially in designating positions. It might be that the only, or senior, resident on the hospital staff would be a "junior" or an "associate resident."

The terms apply to the period of training in the specialty rather than to the executive status of the resident in the specific hospital. The adoption of this uniform nomenclature by all hospitals would help materially in estimating the training of the individual and in comparing the hospital services between different hospitals. The committee recommends that the Academy adopt a resolution recommending the adoption and use of this uniform nomenclature for house officer positions.

In a recent report (*American Journal of Diseases of Children* 51: 389, 1935) the American Board of Pediatrics has called attention to the differences in the quality of training received by the residents in different hospitals. It recommends that a study of the residencies be undertaken by one or more of the Pediatric Societies. Such a survey to be of value would require personal inspection and require considerable time. The committee feels that if sufficient funds and a qualified personnel for the investigation could be obtained, it would be of great service and

would bring the training of residents, our future pediatricians, to a much higher level than exists at present. An incomplete survey or one by questionnaires would be wasted effort. It is estimated that such a study would require approximately $6,000. The committee recommends that the executive committee of the Academy attempt to obtain funds for such a survey to be conducted by the Academy.

A matter that has raised considerable discussion relates to graduate instruction in pediatrics. In our field this is confined almost entirely to the hospital training of residents. It is further complicated by the ruling of the Advisory Board on Medical Specialties and the Council on Medical Education of the American Medical Association that the training of the specialist should include "intensive graduate training in anatomy, physiology, pathology, and the other basic medical sciences which are necessary to the proper understanding of the specialty in question."

The chairman of the committee has by personal interviews or letters made contacts with over fifty pediatricians—chiefly the heads of hospital staffs—in order to obtain opinions as to the scope and character of this basic training and as to how it should be given. As the matter is too involved and too complicated to be discussed in this report, the committee has asked for a discussion of these topics to be held in connection with this meeting of the Academy where the various viewpoints may be presented and discussed.

Borden S. Veeder, Chairman
Thomas B. Cooley — *Jay I. Durand*
James L. Gamble — *Henry F. Helmholz*
A. Graeme Mitchell — *William A. Mulherin*
D. Lesesne Smith

Report of the Committee on Nursing Education

The Committee on Nursing Education has been working toward admitting nurses into nursing schools for training for three to six months. They have been able to accomplish this in at least one institution in San Francisco and have followed the plan which is worked out far more completely in the Children's Hospital in Cincinnati.

William Palmer Lucas, Chairman
Stanley Gibson
William A. Mulherin
Charles A. Weymuller

Report of the Committee on Revision of the Pharmacopoeia.

The Committee on Revision of the Pharmacopoeia proposes the following outline of its work for the coming year:

1. To determine by means of a survey conducted along the line of pediatric literature, a study of the *United States Pharmacopoeia* and *The National Formulary;* pediatric recognition.
   a. Biologic and bacteriologic products.
   b. Chemicals
   c. Nonchemicals or galenicals.
   A compilation of all data referring to the above groups, with source of information, bibliographic reference, etc.
2. A study of vehicles and other means of administering therapeutic remedies, with the thought of improving unpleasant tasting remedies for use in infants and children.

3. A study of those remedial agents considered of pediatric importance and necessity (both official and unofficial) with the thought of improving their status and their retention or deletion, or inclusion (as the case may be) for the *United States Pharmacopoeia* XII and *The National Formulary* VII (1940).
4. A study of posologic standards for infants and children (so far as is possible) for the more commonly employed pediatric remedies, arranged according to age groups (body weights) i.e., for the periods of infancy, preschool and school ages.
5. A concerted effort to eliminate the pernicious practice of prescribing proprietary remedies when equally efficient official preparations can be employed.
6. Restriction in the sale of remedial agents considered potentially dangerous to infants and children over the drug counter, or otherwise, without the written order of a physician.
7. The publication and adoption of a standard compendium and formulary containing official and unofficial remedies of pediatric importance and necessity, with definite indications for the use of such drugs, method of administration and doses. This manual should be prepared to meet the needs of pediatrists and those general practitioners who treat children. It is suggested that a manual of this type should be available to supplement the *United States Pharmacopoeia* and *The National Formulary*, but in no way intended to replace either text. The compendium and formulary should be prepared entirely by your committee and such individuals, or others whom your committee may select should be sponsored by the American Academy of Pediatrics.

On vote of the Executive Board Dr. Bernard Fantus was requested to act on this committee and has kindly consented.

Isaac A. Abt, Chairman
William N. Bradley
Bernard Fantus
Louis W. Sauer

Report of the Council for Pediatric Research of the American Academy of Pediatrics

In June, 1935, the American Academy of Pediatrics accepted the Report of the Committee on Clinical Investigation and Scientific Research. In this report it was pointed out that the present tendency in the acceptance of funds could be successful only if the distribution of funds for research projects is carried out according to high ideals and to our best traditions without regard to consideration of personal friendship, geography, or medical politics.

The committee recognized the advancement of science and research in industrial and pharmaceutical laboratories, and stated it would be unfortunate if such research just now did not proceed hand in hand with the fundamental scientific investigations in university laboratories and clinics.

In order to carry out these objectives the president of the American Academy of Pediatrics appointed the following committee to be known as the Committee on Allocation of Research Funds:

Dr. Kenneth B. Blackfan, Chairman
Dr. Thomas B. Cooley
Dr. Alexis F. Hartmann
Dr. Irvine McQuarrie
Dr. Fritz B. Talbot, Secretary.

The Committee on the Allocation of Research Funds met in Boston, November 6, 1935. All members were present.

According to the consensus, it was decided that the objectives of the Committee should be defined as follows:

"It shall be the purpose of the Committee on Allocation of Research Funds to promote and coordinate research by whatever means medical and biologic science offers in the solution of problems which will benefit the health and welfare of children."

At this meeting tentative plans and policies were drawn up and submitted to the Executive Board which met in Chicago on Sunday, December 1, 1935.

After due consideration the following resolutions were approved:

1. That the Committee on Allocation of Research Funds shall be reconstituted as the Council for Pediatric Research of the American Academy of Pediatrics.
2. That it would welcome the endorsement of this Council by the American Pediatric Society.
3. That this Council shall consist of five or more members.
4. That one member and not more than one each year shall retire.
   (a) That retiring age of each member shall be not greater than seventy years.
   (b) Any retiring member is eligible to reelection.
5. That it is the sense of the Executive Board that as soon as possible the council shall be organized as an autonomous unit within the Academy.
6. That the Academy of Pediatrics lend the Committee on Allocation of Research Funds sufficient funds to start with as was done in the case of the American Board of Pediatrics.

Since this time the committee has been actively at work and now wishes to report progress. It is their feeling inasmuch as this movement is so novel and so important for research in the future that development of plans and policies requires further study. It is the consensus of the committee that the setting up of the Pediatric Research Council serves an important purpose from the point of view of directing monies for pediatric research in a manner which at the present time is not possible with the organizations already interested in this work. They also believe that the Research Council should not be hasty in formulating final plans and that much thought should be given to plans and policies under which the council serves in order to avoid falling into ways which would obstruct the high ideals which the Academy of Pediatrics is trying to develop.

Kenneth Blackfan, Chairman
Thomas B. Cooley
Alexis F. Hartmann
Irvine McQuarrie
Fritz B. Talbot, Secretary

Progress Report of the Committee on School Health and School Health Education

The part of the physician in the development of school health programs has been to a considerable degree brought into disrepute because the *character* of the examination and the advice regarding the health needs of the pupils have *not* met the highest standards of medical service. From the surveys of school medical service and the experience of physicians engaged in this work, it is obvious that the *funds* provided, the *time* allotted to the physician, and the limited clerical and nurse *assistance* provided make it impossible for more than a very few school districts to provide an *adequate appraisement* of physical status, thorough medical examinations, and advice as to the health needs of every school child as frequently as is usually attempted.

It is the opinion of your committee that school children should have *individual preventive* medical service, health examinations, and health supervision, and that this service is not being encouraged in many situations. School children and parents are being wrongly educated because they have been led to expect that this modern form of service can and should be provided in school. There is need for the recognition of the fact that such service cannot be rendered effectively on a wholesale basis. Both the school authorities and the public have demanded a free service in school without taking the advice of qualified representatives of the medical profession as to the *cost and time* required for rendering this service on a basis of satisfactory medical standards. The result is too often rapid inspections by a physician, too little observation for health disorders by teachers and nurses, and insufficient time given by the physician to the cases that demand follow-up.

We agree with the Committee on Medical Service of the White House Conference as to the limited applicability of school examinations and the importance of complete, systematic, serial assessment of the individual healthy child and guidance in the further nurture and training of the child by the medical practitioner.

We believe that the public should be educated as to the importance of preventive medical service by the medical practitioner and that this can never be accomplished if we allow school authorities to lead the public to expect that the school can and does provide an adequate health examination at public expense. Therefore, every school should make clear to its patrons the extent of the medical service provided and its limitations.

To allow school authorities and the public to assume that the school does make or can make a more thorough assessment of the child than is possible leads many parents to a false sense of security; or when they discover some of the limitations of the school medical service, they are inclined to conclude that the service is worthless or they disregard the advice rendered by the school. In this decision they are again in error. Whatever service is rendered by the school, its values and limitations should be clearly stated to parents and to the older children in the interest of sound education as to the values of complete, systematic, serial appraisal of the child and preventive medical service by the medical practitioner. School superintendents, boards of education, and all school authorities should recognize that the responsibility for the character of medical service should be borne by the medical profession and that they should rely upon the profession for a definite statement of the character of the school examinations and inspections, their values and limitations. Likewise school authorities should look to the medical profession for guidance in determining what examinations and inspections should be provided in order that the maximum of protection and education may be available with a given budget and to avoid wasted effort upon hurried examinations and insufficient time for advising the nurse, teachers, and children regarding health needs.

In order to avoid medical neglect of children suffering from definite physical handicaps, the recommendations to parents as to the importance of medical attention should be convincing and based on sufficient knowledge of the case to render sound medical judgments. The school examinations should lead to the securing of the necessary care and treatment of physical defects and therefore, the examination should provide for sufficient time and study of the cases to permit sound medical judgments and recommendations to parents as to the importance of medical attention of each case selected for follow-up.

The Committee on School Health and School Health Education reports the endorsement of three papers of special merit dealing with subjects related to school health work. Two of these were published in *The Journal of Pediatrics* of the American Academy of Pediatrics and one elsewhere. These papers are:

"Forty Years of School Medical Inspection" by Harold H. Mitchell, M.D., J. PEDIAT., Vol. 7, No. 5, November, 1935, p. 714.

"The Physician's View of Health Examination" by Julian D. Boyd, M.D., J. PEDIAT., Vol. 7, No. 6, December, 1935, p. 735.

"Health of the School Child" by W. W. Bauer, M.D., Education, December, 1933.

In addition to these the report on "School Medical Inspection in the United States of America" by James F. Rogers, M.D., Dr. P. H., gives statistics recently collected on many subjects outlined in the tentative study program prepared by this committee for the current year and to be urged for next year also.

The Committee recommends the study of Dr. Rogers' special report (published by Department of Interior of U. S., Bureau of Education) and also the report of the British School Medical System especially as to prevalence of similar defects as reported in England and in the United States—adenoids and tonsils are especially worthy of comparison.

One is impressed with the fact that the close coordination of other governmental services with the British School Medical Service provides better organized effort and integration, and removal of clothing is possible in England, but one might raise the question as to whether the parent is in as complete control of the child's care and treatment as in this country. This appears to be an important consideration which cannot be disregarded in a democratic country under the plea of efficiency.

One must be acquainted with the British medical practice schemes and the British school medical inspection system if he is to discuss intelligently some of the proposals now being made in this country by certain small groups of wealthy and influential persons who advocate methods of practice in this country which are essentially British, and which, according to the views of many American medical leaders, are quite unsuited and unadaptable to American conditions and needs.

The continuation of the study is urged.

LeRoy A. Wilkes, Chairman
Harold H. Mitchell, Secretary
Rockwell M. Kempton
M. Hines Roberts
A. Clement Silverman
Clifford D. Sweet

STUDY PROGRAM OF THE COMMITTEE ON SCHOOL HEALTH AND SCHOOL HEALTH EDUCATION

(Our recommendation is that the study be continued.)

A. *Purpose:* The purpose of this study is primarily to learn what the physicians especially interested in the practice of pediatrics know about the health work now conducted in the public schools; what they believe should be retained or rejected; and what they recommend for the immediate future as a result of this study.

B. *Plan and Scope of Study:* The Plan.—Specific portions of the study are delegated to the various members of the Committee, according to their special fitness. These contributions will be gathered and the entire tentative report will be outlined and a copy sent to each member of the committee for criticism and suggestions. The study will then be revamped and rewritten and submitted to the American Academy of Pediatrics for criticism. The scope shall cover only those considerations which affect the child sufficiently to indicate the economy and wisdom of providing medical supervision and care by the family physician with possible contributions by well-selected physicians in the school as warranted, and where the economic value of such contributions can be defended as proper services for a tax-supported public function.

I. What is the physical status of the children in our public schools?

Criteria:

1. What per cent of absences are due to acute illnesses?
   What per cent of absences are due to communicable diseases?
   What per cent of absences are due to chronic diseases?
   a. Tuberculosis, cardiac, renal, malnutrition.
2. What kind and percentage of significant physical defects handicap the child in school progress?
3. What kind and percentage of functional handicaps interfere, in part at least, with academic progress or require undue physical and mental effort on the part of the child in order to keep up with his classmates?

II. What is now being done in schools to discover or minimize these handicaps?

1. Increased interest in the physical condition of each child (criteria used), as this interferes with his education; improved construction and sanitation of school buildings; better understanding of study and rest periods—their duration and relation one to the other; school administrations; their understanding of physiologic needs, fatigue and recuperative abilities in children; school-teachers.
2. Systematic physical tests and supervision made by competent physicians, nurses, dentists, and physical education teachers, and a clear declaration of the proper scope of function of each.
3. Follow-up efforts and plans which will not only record defects but educate parents as to their significance and how they can be cured or helped through medical service available through their family physician or upon his recommendation.

III. What should be done, how and by whom?

1. Would preschool age supervision by the family physician and the correction of habits, defects, dysfunctions, diseases, be most effective or should it be an organized procedure conducted by the medical society or other health agency?
2. Should physical qualifications and classification (endurance) be set up for entrance to schools?
3. Are special classes in schools justified and economical?
4. Do present methods of follow-up secure the needed improvement?

IV. Education in health conservation.

1. General—by teachers.
2. Urgent (contagion)—by public health nurses.
3. Individual needs—by physicians.

V. Analysis of Special Studies made and conclusions reached.

A. C. H. A.—Five-year analysis and survey.
Metropolitan study.
Harold H. Mitchell, M.D.—Freeport and elsewhere.
James F. Rogers, M.D., Dr. P. H., U. S. Office of Education, Department of Interior, Washington, D. C.
National studies.
Private schools.
Malnutrition.
London County Council—Annual Report of the Council, 1934.
Public Health.

Guiding Principles Recognized.—The medical society should be consulted *in the planning* of Community Health Programs—not *after* the final plans have been made.

The approved plans, schedules, and policies which will govern the conduct of all workers in community projects should be adopted and medical service must be supervised by competent physicians.

The integration of the ideas and activities of those existing agencies whose functions are effective and extensive in their application is an essential part of the program.

The less important agencies should supplement their work under the direction of other workers.

The *plan* and *conduct* of the medical service and supervision thereof are essentially the job of the medical profession. The place in which such services are rendered is of less significance provided that the equipment and quarters are such as adequately meet the needs of the medical examiners.

To aid in the development of proper procedure in the schools, the committee recommends that articles selected or prepared to clarify any part of the aims and procedures in school health programs conducted in schools, or which may be conducted in schools in the future, be printed, with the approval of the School Health Study Committee of the American Academy of Pediatrics in the *Journal of Pediatrics*, if accepted by the Editorial Board of the Journal. Two such articles have already been accepted, "Forty Years of School Medical Inspection" by Harold H. Mitchell," and "The Physician's View of Health Examinations" by Julian D. Boyd.

Recommendations of the Nominating Committee

The following recommendations were made:

For vice president (president-elect):
Dr. Philip Van Ingen, New York, New York.

For chairman of Region II:
Dr. Edward C. Mitchell, Memphis, Tenn.

Thomas B. Cooley, Chairman,
Joseph B. Bilderback,
Edgar P. Copeland,
Hugh Leslie Moore.

No reports from the following:

Committee on Cooperation with Medical Groups and Societies.
Committee on Mental Hygiene.

News and Notes

The Annual Meeting of the **American Board of Pediatrics** was held in Kansas City, May 9 and 10, 1936. All directors and officers were reelected for the ensuing year. Dr. Frank Gengenbach was reappointed as a member of the Board from the **Section on Pediatrics of the American Medical Association.**

The Board has found it necessary to increase the number of examiners and voted to add a group of examiners who shall act as members of the Board for purposes of examination. Dr. T. B. Cooley, Dr. Horton Casparis, and Dr. W. W. Belford were appointed at this meeting.

A total of 224 certificates was granted by examination and on record during the past year.

The fee for examination and certification after September 30, 1936, has been placed at $30. Attention has previously been called to the fact that the fee of $20 now charged by the Board of Pediatrics is much lower than that of the Boards for other special fields. Experience gained in the past two years has shown that an increase in the fee is necessary to meet the expenses of the Board. The increased fee is still lower than that charged by other boards (e. g., Otolaryngology, $50; Dermatology, $35; Ophthalmology, $50).

The following have been certified by the **American Board of Pediatrics** since the last report. None of these applicants were examined.

John William Amesse, Denver, Colo.
Maurice Lamm Blatt, Chicago, Ill.
Jules M. Brady, St. Louis, Mo.
M. Bernard Brahdy, Mt. Vernon, N. Y.
Chester R. Brown, Arlington, N. J.
George Walter Caldwell, New York, N. Y.
D. M. Cowie, Ann Arbor, Mich.
George Martin Guest, Cincinnati, Ohio
Lewis Webb Hill, Boston, Mass.
William W. Howell, Boston, Mass.
Archibald L. Hoyne, Chicago, Ill.
Herbert F. Jackson, New York, N. Y.
Mildred Wicker (Jackson), Oyster Bay, N. Y.
George Kahn, Boston, Mass.
John S. McDavid, Oak Park, Ill.
Morley D. McNeal, Highland Park, Ill.
William Diuguid Mims, Memphis, Tenn.
Dorothea May Moore, Cambridge, Mass.
Karlton G. Percy, Boston, Mass.
Bret Ratner, New York, N. Y.
Aldert Smedes Root, Raleigh, N. C.
James Earl Russell, Jr., Denver, Colo.
D. Lesesne Smith, Sr., Spartanburg, S. C.
Chester A. Stewart, Minneapolis, Minn.
Thomas Durland Van Orden, New York, N. Y.
Arthur E. Wade, Seattle, Wash.
John Zahorsky, St. Louis, Mo.

The **International Congress for the Protection of Infancy** will meet in Rome during the first week of October, 1936, immediately after the International Pediatric Congress.

The annual meeting of the **Louisiana State Pediatric Society** was held at Lake Charles on April 27, 1936.

The following officers were elected for 1935-1936: Dr. Edwin A. Socola, Chaille Building, New Orleans, La., president; Dr. Clarence H. Webb, Medical Arts Building, Shreveport, La., vice president; Dr. Ruth G. Aléman, 1407 S. Carrollton Avenue, New Orleans, La., secretary-treasurer.

The guest speaker was Dr. Walter D. Brown, of Beaumont, Texas. He spoke on the "Use of Adult Blood in Whooping Cough."

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